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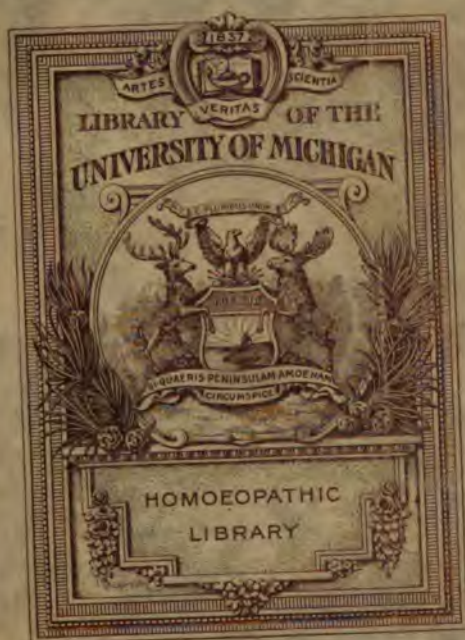
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CONTRIBUTORS TO VOLUME II

ABRAHAMSON, ISADOR, M.D.
CAMP, C. D., M.D.
CASAMAJOR, LOUIS, A.M., M.D.
HALSTEAD, ALBERT E., M.D.
HOLMES, GORDON, M.D., M.R.C.P.
HOPPE, HERMAN H., M.D.
JELLIFFE, SMITH ELY, A.M., M.D., Ph.D.
MALONEY, WILLIAM J. M. A., M.D., F.R.S. (Edin.).
MUNSON, J. F., A.B., M.D.
NICHOLS, HENRY J., M.A., M.D.
RUSSEL, COLIN K., B.A., M.D.
SCHWAB, SIDNEY I., M.D.
SCRIPTURE, E. W., M.D. (Munich), Ph.D. (Leipzig).
SHERREN, JAMES A., F.R.S.C. (England).
TILNEY, FREDERICK, A.M., M.D., Ph.D.
VAUGHAN, ROGER T., M.D.
WILSON, S. A. KINNIER, M.D., B.Sc., M.R.C.P. (Lond.).

THE
MODERN TREATMENT
OF
NERVOUS AND MENTAL
DISEASES

BY AMERICAN AND BRITISH AUTHORS

EDITED BY

WILLIAM A. WHITE, M.D.

SUPERINTENDENT OF THE GOVERNMENT HOSPITAL FOR THE INSANE, WASHINGTON, D. C.;
PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE GEORGETOWN UNIVERSITY;
PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE GEORGE WASHINGTON UNIVERSITY; LECTURER ON MENTAL DISEASES IN THE
U. S. ARMY AND U. S. NAVY MEDICAL SCHOOL,
WASHINGTON, D. C.

AND

SMITH ELY JELLIFFE, A.M., M.D., PH.D.

ADJUNCT PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM IN THE POST-GRADUATE
MEDICAL SCHOOL AND HOSPITAL; VISITING NEUROLOGIST TO THE CITY HOSPITAL; CONSULTING NEUROLOGIST TO THE MANHATTAN STATE HOSPITAL, NEW YORK, N. Y.

VOLUME II

ILLUSTRATED



LEA & FEBIGER
PHILADELPHIA AND NEW YORK
1913

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CONTRIBUTORS TO VOLUME II

ISADOR ABRAHAMSON, M.D.,

Neurologist to the Montefiore Home; Associate Neurologist to the Mount Sinai Hospital, and Neurologist to the Mount Sinai Dispensary, New York, N. Y.

C. D. CAMP, M.D.,

Clinical Professor of Diseases of the Nervous System in the Medical Department of the University of Michigan, Ann Arbor, Mich.

LOUIS CASAMAJOR, A.M., M.D.,

Chief of Clinic of the New York Neurological Institute; Assistant Neurologist to the City Hospital, New York, N. Y.

ALBERT E. HALSTEAD, M.D.,

Professor of Surgery in the Northwestern University Medical School; Attending Surgeon and Chief of Staff, St. Luke's Hospital; Attending Surgeon Cook County Hospital, Chicago, Ill.

GORDON HOLMES, M.D., M.R.C.P.,

Assistant Physician to the National Hospital for the Paralyzed and Epileptic, London; Physician to Out-Patients, Seamen's Hospital, London, England.

HERMAN H. HOPPE, M.D.,

Professor of Neurology in the Medical Department, University of Cincinnati; Neurologist to the Cincinnati Hospital, the Good Samaritan Hospital, and the Good Samaritan Annex, Cincinnati, Ohio.

SMITH ELY JELLIFFE, A.M., M.D., PH.D.,

Adjunct Professor of Diseases of the Mind and Nervous System in the Post-graduate Medical School and Hospital; Visiting Neurologist to the City Hospital; Consulting Neurologist to the Manhattan State Hospital, New York, N. Y.

WILLIAM J. M. A. MALONEY, M.D., F.R.S. (EDINBURGH),

Adjunct Professor of Neurology in the Post-graduate Medical School; Attending Neurologist, Neurological Hospital; Assistant Attending Neurologist to the Post-graduate Hospital, New York, N. Y.

J. F. MUNSON, A.B., M.D.,

Resident Pathologist in the Craig Colony for Epileptics, Sonyea, N. Y.

HENRY J. NICHOLS, M.A., M.D.,

Assistant Instructor in Pathology and Bacteriology in the Army Medical School, Washington, D. C.; Captain Medical Corps, United States Army.

COLIN K. RUSSEL, B.A., M.D.,

Demonstrator in Medicine in the McGill University; Neurologist to the Royal Victoria Hospital, Montreal, Canada.

(v)

SIDNEY I. SCHWAB, M.D.,

Visiting Neurologist to the St. Louis City Hospital, St. Louis, Mo.

E. W. SCRIPTURE, M.D. (MUNICH), Ph.D. (LEIPZIG),

Associate in Psychiatry in the Columbia University; Director of the Research Laboratory of Neurology in the Vanderbilt Clinic, New York, N. Y.

JAMES A. SHERREN, F.R.S.C. (ENGLAND),

Surgeon to the London Hospital; Surgeon to the Poplar Hospital for Accidents; formerly Erasmus Wilson Lecturer in the Royal College of Surgeons, London, England.

FREDERICK TILNEY, A.M., M.D., Ph.D.,

Associate in Anatomy in the Columbia University; Lecturer in Neurology in the Long Island College Hospital; Attending Neurologist to Seney, Coney Island, Bushwick, and Samaritan Hospitals; Assistant Visiting Neurologist to the Kings County Hospital, Brooklyn, N. Y.

ROGER T. VAUGHAN, M.D.,

Instructor in Surgery in the College of Physicians and Surgeons; Adjunct Surgeon to St. Luke's Hospital, Chicago, Ill.

S. A. KINNIER WILSON, M.D., B.Sc., M.R.C.P. (LONDON),

Assistant Physician and Lecturer in the Medical School, Westminster Hospital; Assistant Physician and Pathologist, National Hospital for the Paralyzed and Epileptic, London, England.

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NERVOUS AND MENTAL DISEASES

CHAPTER I

TREATMENT OF THE NEURALGIAS AND NEURITIDES

By SIDNEY I. SCHWAB, M.D.

FROM the standpoint of treatment the neuralgias and neuritides may be taken together, as they present to the neurologist almost identical problems. They are disturbances in the function of the peripheral nervous mechanism, having to do with pain and abnormalities of sensation, and with motor anomalies, all the way from slight variations of voluntary movement to complete paralysis; and, again, with various combinations of both.

Pathologically, there is very likely a common basis. The two together may be taken as an expression of an inflammatory process in the peripheral nerve structure, which is sufficiently crude in its histological manifestations to be the subject of microscopic demonstration in the case of neuritis and to escape it in a majority of the neuralgias. The term inflammation must be taken in this instance in a very crude sense, implying anything from a marked infiltration of the myelin sheath and surrounding perineural and connective-tissue structures with the products of an inflammatory process—plasma cells, round cells, bacterial invasion, etc.—and gross mechanical alterations due to crushing, tearing, and cutting of the nerves, to finer changes in the intimate structure of the nerve which escape microscopic demonstration. It must be thought of as a change differing very little in some instances from that produced by the normal activity of the nerve, whatever that may be considered to be.

It is this broad field of pathological phenomena and the correspondingly involved therapeutic problem that give to the neuritis and neuralgias their great interest and importance. Etiologically, the two may be considered as intimately connected, for a common causative incident may produce in the one case a neuralgia and in the other a neuritis.

Owing to this complexity of pathological changes, it is easily seen that there must be somewhere a limited territory in which the neuritides and the neuralgias cannot be differentiated. Here slight changes pass over into negative findings, and the symptoms do not follow as accurately as the causes which underlie them might imply. In this field a differential diagnosis between the two conditions is almost

impossible and the clinical manifestations are as completely a matter of doubt. It is in this place, too, that the pure types of neuralgias lose something of their characteristic clinical manifestations and the neuritides, on the other hand, take on something of the neuralgic manifestations. Therefore, the two processes are intimately related; at either extreme the types are sharply differentiated with a tendency to approach, as has been said, the *Grenzgebiet*, where the pathological, the etiological, and the therapeutic problems are practically identical.

There are three important causative factors at work in the production of neuralgic and neuritic manifestations:

1. There are the various types or subvarieties of mechanically acting processes, such as crushing, pressure, tearing, cutting, etc. These produce destruction of the peripheral nerve tissue as such.

2. There are changes produced in the nerves due to poisons of an absorbable sort, introduced from without, such as lead, alcohol, arsenic, etc.

3. There are changes produced by the action of organisms or their products by poisons of an endogenous nature, such as diabetes, the so-called auto-intoxication processes, and others of a similar nature.

It is to be understood that this classification is made solely to facilitate therapeutic ends and does not rest upon any more significant differentiation.

NEURALGIAS

Primarily, neuralgia means pain, or a painful sensation of a certain kind, limited to the sensory distribution of a peripheral nerve, or nerves, or referred to the surface distribution, pressure upon which causes a typical, though not severe, neuralgic pain. Neuralgias are the sensory anomalies of the motor disturbance, to which the term neuritis is given; that is, a similar process limited chiefly to sensory nerves is spoken of as neuralgia, while the identical process in a mixed motor and sensory disturbance is regarded as neuritis. This is the clinical differentiation, but, as a matter of experience, the neuritides are the result of gross changes in the peripheral nerve, which frequently can be demonstrated under the microscope, whereas in neuralgias just as frequently the microscopic changes bear no definite relation to the clinical symptoms. In the case of a mixed nerve, where, in addition to the motor disturbance, there is at the same time pain, it is better to regard the process as neuritic, rather than neuralgic.

The therapeutic problem in all neuralgias is primarily the temporary, or permanent, relief of the chief symptom, pain. To do this necessarily implies an understanding of the producing agent of the pain and the underlying factors at work. To accomplish this, two sets of facts must be obtained, one having to do with the cause locally at work at the point of pain production, and the other having to do with the underlying causes, if any, which are the activating agencies back of the local area of disturbance. To illustrate: A neuralgia in the lumbar region may be due to arthritis, or osteitic changes in the spinal column. This

causes a mechanically produced pain by pressure on the posterior nerve roots. The underlying causes in such an instance are the processes causing the arthritic changes, which are probably of a metabolic sort. The neuralgic manifestations are due purely to the locally effective pressure on the posterior nerve roots and are only an expression of a much more general abnormal process.

A complete therapy would necessarily imply treatment of both the local and the general causes. The one, perhaps, by mechanical means, and the other by measures adequately designed to counteract the general process. It is possible, however, only in a few instances to do this, but it is well to keep in mind the larger therapeutic problem so frequently involved.

The neuralgias, for purposes of therapeutic attack, are capable of various sorts of classification, depending upon the point of view—regional, etiological, pathological, etc. No one classification with our present knowledge can be regarded as at all satisfactory, and that classification which presents a certain convenience for descriptive exposition will be here adopted—in the main, it will be a regional classification.

For purposes of description, the term neuralgia may be regarded as somewhat elastic, as some of the so-called pain symptoms given a formal terminal designation are really not neuralgic according to a strict interpretation of the meaning of that term.

Quintus Neuralgia.—The most important cranial nerve which becomes the seat of neuralgic pain is the trigeminus. This is a prototype of the classical neuralgias and has been taken for a long time as the best example of the clinical picture of neuralgia—not only on account of the typical character of the pain, but because its anatomical distribution has brought out so sharply this feature of a typical neuralgia.

Types.—Therapeutically considered, the neuralgias of the fifth nerve present two distinct types to be regarded chiefly from the point of view of intensity, continuousness, and the influence on the life, activity and capacity of the patient. This distinction is purely one based upon the severity of the symptoms. The symptomatic manifestations of trigeminal neuralgia cover a wide field, from the slight twinge characteristic of pain felt somewhere in the distribution of the fifth nerve, due to some slight irritation, to the dramatic picture of intense suffering seen in the extreme *tic douloureux*. It is thus seen that neuralgia of the fifth nerve may be so slight as almost to escape attention; or, again, so severe that the patient cannot possibly live, or be active in the presence of the intense pains. It can, therefore, be easily seen that therapeutically an effort at relief must be largely influenced by the severity of the symptoms. In order to form a basis for a proper therapy for trigeminal neuralgia, it is necessary to have some idea of the anatomical distribution of the sensory portion of the nerve, both in extent and location.

Anatomy and Distribution of Fifth Nerve.—The Gasserian ganglion imbedded in Meckel's cavity is the peripherally lying sensory nucleus

of the fifth nerve, analogous to a posterior root ganglion of a spinal sensory nerve. The axones of the unipolar ganglion cells divide into two branches: One branch is extended peripherally, constituting the peripheral nerve, the course of which will be described later; the other passes centrally toward the pons and enters it on its lateral aspect and runs to the sensory end nucleus of the trigeminus, in close relation to the motor nucleus of the same nerve. The internal fibers then divide into an ascending and descending branch. The ascending branch ends within the sensory nucleus, within the pontine tegmentum; the descending branches can be followed as far as the cervical cord, being gradually lost in the substantia gelatinosa Rolandii which caps the posterior horn.

From the sensory end-nucleus the second neurone arises. The fibers pass toward the midline, giving off collaterals to the nucleus of the facial nerve, cross to the fillet tract of the opposite side, then turn upward and run forward (partly within the medial fillet and partly as a more laterally placed special ascending bundle), and later enter the thalamus with the median fillet. Finally, a third neurone succeeds the second one linking the thalamus with the sensory area of the cortex. Sensory fibers, which pass direct to the cerebellum as constituents of the direct sensory cerebellar tract are also to be considered; further, there are fibers which pass from the sensory end-nucleus to the cerebellum as constituents of the tractus nucleocerebellaris. (Adapted from Villiger.)

The sensory root, upon which the Gasserian ganglion is located, divides below the ganglion into three divisions, of which the first two are entirely sensory. The motor root courses beneath the Gasserian ganglion, and then joins the third division, which thus becomes a mixed nerve.

The first, or ophthalmic, division passes through the sphenoidal fissure into the orbit and supplies the eyeball and lacrymal gland, the conjunctiva (except that of the lower lid), the skin of the forehead and scalp up to the vertex, the mesial part of the skin of the nose, and the mucous membrane of the upper part of the nasal cavity. It also contains efferent pupil-dilating fibers derived from the cervical sympathetic, joining it at the Gasserian ganglion, and going to the iris.

The second, or superior maxillary, division passes through the foramen rotundum across the sphenomaxillary fossa to the infra-orbital canal. In the sphenomaxillary fossa it is connected with Meckel's ganglion, which gives off, with other branches, the Vidian nerve. This latter runs backward to join the facial nerve, the posterior end of the Vidian being named the great superficial petrosal. The superior maxillary division supplies the skin of the upper lip, the side of the nose and adjacent part of the cheek, the lower eyelid and part of the temple. It also supplies the conjunctiva of the lower lid, the upper teeth, the mucous membrane of the upper lip, the upper part of the cheek, upper jaw, uvula, tonsil, nasopharynx, middle ear, and lower part of the nasal cavity.

The third, or inferior maxillary, division is a mixed nerve. It emerges through the foramen ovale. The motor fibers supply the masseter, temporal, both pterygoid, tensor tympani, mylohyoid, and anterior belly of the digastric muscles. The sensory fibers supply the skin of the posterior part of the temple and adjacent part of the pinna, the anterior and upper wall of the external auditory meatus, as far as and including the anterior part of the drum, part of the cheek, the lower lip and chin, also the lower teeth and gums, the tongue (as far back as the circumvallate papillæ), floor of mouth, inner surface of cheek, and salivary glands.

This, in a general way, will give some idea of the widespread area of the fifth nerve and will serve in a measure to explain the great variety of symptoms, both in distribution and quality, which the affection that is here being considered will show.

In addition to the usual symptom of pain which neuralgia of the fifth nerve shows, there is a form in which the clinical picture is somewhat more complicated by the addition of motor symptoms; that is, in the variety known by the name of *tic douloureux*. This may be regarded merely as a measure of the pain intensity, but does not separate the therapy from the other types of severe trigeminal neuralgia. When the combination, however, of spasm and pain is met with it is a sure evidence that we are dealing with one of the severest types of facial neuralgia.

In a general way, all neuralgias of the fifth nerve must be regarded as a manifestation of some known or unknown form of sensory irritation. Whether this is to be located extratrigeminally, or in the distribution of the nerve itself, cannot always be determined. At any rate, the first thing to consider in the presence of neuralgia of the fifth nerve in any degree of severity is to note and discover the possibility of a demonstrable lesion, acting upon the fifth nerve as such.

When the widespread distribution of the fifth nerve is considered, together with its close relationship to bone and bone cavities, the possible etiological factors become at once manifest, and no degree of care is too exacting, which has for its purpose the discovery of some definite cause, particularly such as might act purely mechanically by pressure in any of its manifold varieties. A careful examination of the teeth and gums and of all the sinuses in which the fifth nerve has any possible distribution is among the first considerations, and the correction and modification of abnormal processes if found is of the utmost importance. It might be stated here, however, that even with the correction of the manifestly mechanical causes of irritation, the relief of pain does not necessarily follow, for the reason that the inflammatory process set up in the sensory nerve does not always cease with the removal of the cause. This explains, probably, why the percentage of cures following correction of very obvious defects in the teeth, gums, or sinuses is so low.

Treatment.—In the light of manifestations of trigeminal neuralgia, the treatment should be directed, after the considerations previously

noted, purely to the relief of pain and the measures that are at our command to be used and rejected are largely governed by the severity of the pain manifestations. Of great importance is the fact that in the presence of periodically occurring pain only such analgesics should be used as carry with them no habit-forming tendency and no influence upon the general welfare of the patient and no tendency toward lessening the pain-bearing capacity; therefore, morphine and its various derivatives, or substitutes, should not be used, except in extreme cases, or to tide over the interval leading to surgical measures. When, after the use of more or less prolonged treatment with the milder analgesics, such as aspirin, salicylates, and others belonging to that class, the attacks of neuralgia are not materially lessened, and when the attacks begin to become a more or less serious menace to the patient's comfort, peace of mind, ability to work, or to live out his accustomed life, then relief through surgical treatment must be considered. This statement is, naturally, conditioned upon measures planned for the correction of abnormal processes in the mouth, gums, and cranial sinuses.

Surgical Treatment.—There are at present two distinct surgical procedures in common use. Both are essentially based on the same general idea; that is, they both attempt to cut off from the brain, where the central interpretation area of pain must be located, the peripherally lying segment of the trigeminal nerve where the actual process of pain initiation must be. The two methods include (1) an attack on the Gasserian ganglion itself or the sensory root of the ganglion; and (2) a peripherally lying operation, such as alcohol injection into the three main divisions of the Gasserian ganglion, or at their points of exit through the bones, or the cutting or tearing of peripheral branches as they appear at the foramina of exit of the three main divisions. The latter operation is mentioned here more for the reason of its historical interest than its present-day usage, as the former operation has largely given way to the alcohol procedure.

ALCOHOL INJECTIONS.—The technique of the latter operation has been elaborated in this country chiefly by Patrick and Hecht. It can be carried out successfully after a careful study of the bony landmarks. If care and cleanliness are used the operation is neither difficult nor dangerous. Reference to Chapter XVII by Halstead will enable the reader to grasp the essentials of the operation. Much depends for its success upon an accurate fixing in the operator's mind of the direction and depth to which the needle should be inserted. Inasmuch as the alcoholic degeneration of the nerve is not permanent relief of pain is only temporary. Complete relief, as given by the various writers on the use of this method, averages about nine months. In some cases the operation must be repeated a number of times.

Neuralgias of the first ophthalmic division cannot be treated by this method, on account of the possible danger to the eye.

There is, therefore, in the alcohol injection of the second and lower branches of the trigeminus a ready method of affording almost instan-

taneous relief for the milder and moderately severe grades of trigeminal neuralgia, when the relief to be obtained is not necessarily permanent and when the repetition of the operation, if necessary, offers no particular objection. If we consider that the greater majority of all neuralgias of the fifth nerve belong to this rather milder class, and, further, when it is remembered that neuralgias of this kind show a curious tendency to disappear, or become less pronounced as the patient advances in years, then the alcohol operation seems to be a very practical and efficient means of securing relief. By repeating the operation a few times, the period during which the attacks may be expected to appear can be easily bridged over.

GASSERIAN OPERATION.—For the severest types of trigeminal neuralgia, in which the attacks of pain are so frequent or so destructive to the comfort and peace of mind of the patient as to be almost incompatible with living, relief is so necessary that no recourse to milder measures can be thought of. The danger in this class of cases from the morphine habit is so imminent, and so serious when once the habit is formed, that long before morphine is used to any extent the more serious operation on the ganglion should be considered. If this operation is successfully performed, it is impossible for the patient to ever afterward have any manifestation of pain, in the distribution of the fifth nerve upon the side which has been operated upon, because either the removal of the ganglion itself, or the severing of the sensory root, cuts off absolutely any connection between the brain and the peripheral distribution of the fifth nerve. It has been proved experimentally that there is no regeneration centrally of the sensory branch of the fifth nerve.

An operation on the Gasserian ganglion should, therefore, be advised in two classes of cases—one in which all branches of the facial are involved and the attacks of pain are of the above-described severity, and in cases in which no relief has been obtained by the temporary procedure of alcoholization in its various methods. In the hands of a few skilful operators this operation has become much less dangerous. The mortality is not much greater—perhaps less—than many other life-saving operations. It must not be forgotten that in the presence of neuralgias of the severest form relief is just as essential from the standpoint of life-saving as in many abdominal emergencies, let us say, and, therefore, the operation should be considered much in the same light as a physician would consider a necessary and serious operation for the maintenance of the patient's health and activity. Whether the ganglion is removed entirely, or whether the sensory root alone is severed, are matters of purely technical concern. In either case relief, and permanent relief, is practically sure to follow, and with no necessarily untoward consequences; that is, if the modern technique is carried out with care and skill, in the hands of a surgeon who has a great deal of experience in this sort of work.

In summing up, therefore, the measures that can be directed to counteract the effects of neuralgias of the trigeminus, it is found that for all varieties there are measures at hand which are sufficiently effec-

tive to offer to the patient some certain promise of relief. From the milder analgesics to the Gasserian operation, each adapted rather to the degree of pain and its effect upon the patient than upon qualitative variations, there is a wide variation of therapeutic procedures.

Constitutional Diseases and Quintus Neuralgia.—There are, perhaps, certain constitutional diseases which seem prone to excite neuralgias in the trigeminal distribution. Just how these work is not at present known. At any rate, the patient should be carefully examined in each case for any evidence of constitutional disease. For example, malaria in some cases has been shown to be the probable exciting cause, or, at any rate, quinine has given relief. The neuralgic pain in suspected malarial cases is supposed to take on a certain periodicity comparable to the variations in the symptoms of the malaria itself. In such cases quinine should be tried, even if the blood shows no evidence of the malarial organism. Naturally, syphilis has likewise been regarded as a probable etiological factor in certain cases, and with the well-recognized tendency of syphilis to attack the bones, it is easily understood that direct mechanical effects could be exercised upon the trigeminal branches in their passage from bony canals to the skull, on their way to the exit foramina in the face. The use of mercury in suspected cases of this kind is to be recommended, though it would appear that this type of facial neuralgia is seldom encountered. The Wassermann blood test is useful in clearing up the etiology of certain syphilitic fifth-nerve neuralgias.

The tendency of diabetes to attack the sensory nerves is well known, and neuralgia of the fifth nerve is found once in a while apparently having some relation with this disease. However, the frequency of diabetes attacking the teeth and gums probably accounts for the prevalence of neuralgias of the fifth nerve when found.

A rare condition which causes very typical fifth-nerve neuralgia is tumor of the Gasserian ganglion, or an inflammatory process of the bones in this neighborhood. Both these conditions will exert pressure in a very distinct way and give rise to fairly well-known symptomatic groups. A differential distinguishing point is usually the involvement of the motor division of the fifth nerve. A combination of neuralgic pains in the distribution of the trigeminus, plus involvement of the motor division of the fifth, gives a clinical picture that is not difficult to recognize. Although this condition is comparatively rare, it should be considered, particularly in those cases which do not respond to any of the therapeutic measures which have been touched upon. It is conceivable that pain due to a cerebral tumor might be referred to the distribution of the trigeminus and be masked in the diagnosis by the presence of an ordinary trigeminal neuralgia—in fact, such cases have been described. In all cases of severe and intractable trigeminal neuralgia every effort should be made to exclude all simulating conditions, for it is only then that remedial measures can be directed and intelligently applied in the way that has been described.

Cervico-occipital.—Pain of neuralgic character is at times met with in the distribution of the occipital and cervical nerves. The location of this pain is more definite and characteristic of the typical spasmodic, shooting pain in the distribution of the sensory nerves. It is seldom met with as an isolated symptom group. The pain area is in the back of the head and neck and can only be diagnosticated as neuralgia when all other causes have been rigidly excluded. In all cases of persistent pain in this region, even though the pain is characteristic of neuralgia, tumor of the brain, particularly the posterior fossa, should be carefully excluded. The nerves affected come usually from the first four cervical nerves, and it is impossible to determine in a case of neuralgia in this region whether the process is located in the roots, or in the peripheral trunks. Head's law of reversal of epicritic sensibility may be of service in this particular. A full consideration of this law is found in the chapter by Sherren. A careful examination should be directed toward discovering the presence of arthritic changes in the upper cervical vertebræ, tuberculosis, carcinoma, and particularly metastasis from cancer of the breast.

TREATMENT.—Treatment is, naturally, directed toward the underlying cause, or causes, if any are found to exist—in fact, any cause which mechanically, or toxically, affects the upper cervical roots might produce manifestations of a marked neuralgic character in the peripheral distribution of the nerves which came from these roots. Influenza, malaria, and other infections are sometimes followed by persistent neuralgic pain, and the treatment is logically that of the disease which produced it. In malaria, naturally, the greatest success will be obtained. Syphilitic neuralgias in this area are not infrequent. The lesion is a radiculitis or is in the bony structures. Salvarsan and mercury are indicated here. A persistent neuralgia of the type found in the fifth nerve is a rarity in this sensory distribution and seldom calls for the radical measures so frequently found necessary there.

Vagus Neuralgia.—The question of the possibility of neuralgia of the vagus is still subject to considerable doubt. That the vagus contains sensory fibers is now admitted, but whether we have any symptomatic expression of such a process is questionable. The pain of angina pectoris, gallstone colic, certain forms of tuberculous processes in the pleura and other similar affections, the effects of which would show themselves in the vagal distribution, should theoretically, without doubt, give rise to very definite neuralgia-like symptoms, but as yet we are in no position to consider them definitely as manifestations belonging to this class. They may be considered, more likely, as referred pains in the sense of Head as surface projections of deeper-lying, pain-producing processes.

As far as treatment is concerned, the problem is that of the chief etiological factor at work, and whether the pain symptom is to be considered as neuralgia or not, it will be benefited or not as the original process reacts favorably or the reverse to treatment.

Brachialgia.—Neuralgic pain in the distribution of the brachial plexus is a very common disturbance and is met with about as frequently as trigeminal neuralgia, or ischias. It seems to have increased since the time that influenza has become more or less endemic, following the first great gripe epidemic in the early 90's.

The distinction between brachial neuralgia and neuritis is at times rather difficult to make, but if we limit our conception as far as possible to such typical painful manifestations in the nerves of the brachial plexus unaccompanied by evidence of paralysis, or degenerative changes in the muscles, and other symptoms pointing to a gross process in the peripheral nerves, we can, perhaps, have a sufficiently clear delimitation to furnish us with the basis for the proper therapeutic efforts.

Anatomy and Distribution.—The brachial plexus is made up from the lower four cervical roots and the first and sometimes the second dorsal root, and it can be easily seen, therefore, that the therapeutic problem will always contain within itself the effort to distinguish between the root, the trunk, and the branch lesion. This can frequently be done by consideration of the pain localization, together with the points that are tender to pressure. If the pain is described as lying within the surface distribution of the peripheral nerve, and is limited to that distribution, then the conclusion is justified that we are dealing with a peripheral branch process. If, however, the pain is distributed over an area supplied by several nerves, if limited by the longitudinal distribution rather than according to the extent of the territory involved, the process may be sought either at the roots or in the plexus itself. The problem of localization, whether root, plexus, or a peripheral branch, is important and can be solved by neurological diagnostic methods which are in vogue in the cases of neuritis in the same locality, and will be considered more in detail in the chapter devoted to this subject, because in neuralgia the necessity for accurately distinguishing between a root and plexus lesion is not nearly so vitally important as in the case of the neuritides, where surgical procedures are frequently found necessary, and where the problem of localization is of great consequence.

Symptoms.—The symptomatology is essentially that of neuralgia elsewhere in the nervous system, the pain is characteristic, very often severe, coming on in attacks accompanied by tender points along the course of the nerves. There is a further symptom to be noted; that is, neuralgias of the brachial plexus are accompanied by a certain amount of loss of muscular power, evidently the result of the pain when motion is attempted, together with a feeling of heaviness and paresthesia.

In every case of neuralgia-like pain anywhere in the distribution of the brachial plexus it becomes of primary importance to determine where the process is and what the exciting cause may possibly be. Pain in the arm may frequently be but a part of the clinical picture of a much more extensive process, which may be located in the cervical spine, or shoulder-joints, possibly a tumor at or near these regions.

In doubtful cases an x-ray examination is essential for determining just what may be present. Pain of a distinct neuralgic character may be the earliest manifestation of a tumor of the spinal cord, or an arthritic process in the cervical spine, or a distinct localization of a tabetic, or syphilitic tabetic-like (meningomyelitis-radiculitis) process in the cervical roots. In none of these cases need there be any evidence of a neuritic change whatever, and in the absence of any definitely localized motor symptoms, the assumption of a pure neuralgic process for the time being must be assumed. An important early symptom of cervicæ Potts is a root neurologic pain, referred to the elbow region. In considering the possible etiology of brachial neuralgias this should not be forgotten.

Treatment.—Treatment, therefore, in the first instance should be directed to the causation, if that is in any possible way to be reached by direct therapeutic means.

Like most of the neuralgias, therapeutic measures should be planned to correspond to the severity of the symptoms, their disabling features and their effect upon the individual sufferer, as far as his activity and peace of mind are concerned. For the most severe type, that in which the pain attacks are almost constantly present and when the effect upon the patient is so destructive as to render existence almost impossible, a resection of the posterior root, properly performed, offers a definite, and sometimes permanent, measure of relief. There are certain necessary provisions to be determined before this procedure should be resorted to. First, a sufficient number of posterior roots should be severed, so as to include the total sensory supply of the arm and the unilateral character of the pain should be very clearly demonstrated, for the reason that the crossing of the fibers to the other side in the cord make the bilateral character of pain almost a necessary consequence. In the more moderate varieties of brachial neuralgias, however, no such radical measures are necessary. In those which are caused by disease processes in the cervical vertebræ, relief of pain depends largely upon the success of treatment directed toward the underlying causes, the measure of support from the orthopedic point of view, mobilization, and other like measures. In cases of new growth in the cervical cord, naturally removal of the offending factor is an essential step toward relief of pain. In ordinary neuralgias, however, whatever their cause may be considered to be, toxic or the result of an infection, local measures have an important place, and among them none is of more direct value than hydrotherapeutic measures applied to the seat of the pain in the arm. It is remarkable to observe the almost immediate relief which will follow hot, moist applications properly used. The whole arm should be immersed in hot water for as long a period of time as the patient can conveniently stand it. Following this, the arm should be enveloped in hot moist bags extending over the whole area. This procedure should be repeated a number of times a day, for periods of about fifteen minutes at a time. The milder forms of acute neuralgic attacks can frequently be successfully treated in this way, or, at least, so far

relieved that the pain ceases to be of any considerable importance. The value of the salicylates, or aspirin, or similar preparations cannot be disputed, but their long-continued use soon exhausts their power of relief, and inasmuch as they act largely as pure analgesics, they soon cease to be effective, and their consequences in the way of gastric and other symptoms are not very agreeable.

The use of electric light, for the application of dry heat, long immersion in hot water, and other physical methods are naturally of some benefit. From electricity not much can be expected, even from what may be called the psychical effect, but with the exception of its use in the acute stages, no particular objection against it can be set down.

A word should be said here against the indiscriminate use of massage, particularly in the initial period of neuralgic attacks. In the presence of tender peripheral nerves even very mild massage is contraindicated. Rest is an advisable method of treatment, and sometimes splinting the whole arm in a well-arranged bandage serves to give the desired relief.

Intercostal Neuralgia.—This is a very common form of neuralgia, and probably is more frequently diagnosticated than its occurrence might justify. Intercostal neuralgia is a neuralgic pain along the course of any of the intercostal nerves, or in several of them. As in the case of brachialgia, the process may be regarded as strictly peripheral in expression, or due to a root process involving several of the peripheral segments. An important thing about intercostal neuralgia is the diagnosis. Etiologically, there are so many possible factors that might produce pain in this distribution that care must be taken to exclude processes which should be treated of themselves. Therapeutically, this important point is easily admitted. Tuberculosis, pleurisy, processes about the posterior nerve roots, tumors of the breast, mediastinum, aneurysm, and various other general conditions may cause an intercostal neuralgia as well as anemia, pectoral conditions, syphilis, etc.

Of particular importance are the neuralgic symptoms found in herpes zoster, which have all the characteristics of a root neuralgia. As might be expected, these are lesions in the posterior root ganglion.

A frequent form of neuralgia is found in pregnant women, in the early months of pregnancy, when the breast first becomes enlarged. This is particularly stubborn and presents difficulties in treatment, but with the establishment of the flow of the milk and the regular filling and emptying of the breast the pain disappears. What has been said in regard to treatment of the other forms of neuralgia would be true here. Mobilizing the chest wall by adhesive strapping and by tight bandaging can often be tried successfully, especially so in the exhaustive forms.

In intercostal neuralgia particularly a diagnosis of the underlying causes is of the utmost importance, because among the causes are many that offer a chance for successful treatment, or the effects may be made less annoying by intelligent treatment.

In all cases of herpes zoster accompanied by neuralgic pain of the root variety, care must be taken to discover if the zoster symptoms are not part of a process in the spinal cord or its roots of a degenerative kind. Tabetic-like lesions may show themselves first by the outbreak of a crop of herpes along the intercostal distribution, which may be for a time the only definite symptom present.

Mastodynia.—This is a neuralgic pain which has its location in the distribution of the third and fifth dorsal segments, and, at times, from the lower cervical segments. It is rarely seen in men, and most frequently in women of the so-called neurasthenic type. It is a true neuralgia and, naturally, all organic causes, such as tumor and other processes before alluded to, should be excluded. The true mastodynia should be differentiated from the so-called hysterical breast, described by Charcot under the term "sein hysterique." In the latter there exists a hypesthesia of the skin of the mammæ, which can easily be demonstrated, and is subject to the usual clinical tests for hysterical sensory disturbance.

Apart from the care necessary to exclude organic conditions, the treatment of mastodynia needs nothing special beyond the usual measures which have been described in the other neuralgias.

Neuralgia of Lumbar Plexus.—Neuralgias of the lumbar plexus include in their distribution the sensory nerves which spring from the fifth lumbar segment. The neuralgias here obey the same laws of distribution seen in the other plexus types. There are the root, the plexus, and the peripheral distributions.

Neuralgic pains here are rather common, but they seldom have as definite a symptomatology as some of the other types of plexus neuralgias. It is evident that the sense of accurate projective localization is less markedly developed than in the brachial types for instance.

Neuralgias of the gluteal region are fairly common. Orchidynia should be mentioned particularly, although the pain here is scarcely to be regarded as typically neuralgic in character. Paresthetic sensations are more frequently met with than typical neuralgic manifestations; they are very common as manifestations of organic processes in the genital tract in both men and women. The gluteal pain, so-called backache, found frequently as a part of the symptoms of uterine displacements, should scarcely be included in a description of neuralgias, though at times their symptomatic manifestations are strikingly similar.

Therapeutically little additional can be said for the neuralgias here. The possibility of organically produced pain is of particular importance, especially in all cases following traumas of various kinds. Some of the painful sensations incident to traumatic neuroses of various sorts and produced in various ways are strikingly like neuralgia and it is necessary to differentiate carefully between the two. Neuralgic manifestations depending upon a functional disturbance in a given nerve distribution are sometimes met with. They are pains that are largely emotional in character and projected mentally to a certain peripheral

distribution. There is a very typical neuralgic-like pain in the gluteal region, in the distribution of the internal cutaneous nerve, following traumatism, as a part of the picture of the traumatic neuroses.

Ischias or Sciatica.—Pain in the sciatic nerve, sciatica, or ischias is one of the very common forms of neuralgia. It is almost as frequently found as trigeminal neuralgia, and its consideration gives rise to many diagnostic errors. The distribution of the pain lies in the distribution of the upper roots of the sacral plexus. Symptomatically, the pain is fairly characteristic of the typical neuralgias. The sciatic nerve, owing to its exposed position and to its long course, is easily palpable and its characteristic tender points can be demonstrated without difficulty, especially at the sciatic foramen and in the popliteal space. The hyperextended leg flexed toward the body causes a very characteristic pain, shooting from the exit foramen in the sacrum down toward the heel, with marked coincident muscular spasm (Lasague's sign). This is almost a certain proof that the neuralgia exists in that nerve.

There are also attacks of pain in the sciatic nerve of another type. Here the pain comes in sudden attacks, with, apparently, free intervals between them. These bear no definite relation to the position in which the leg happens to be held. In some patients there is distinct relief on walking and on making other movements, whereas in others total rest is a necessity if pain is to be avoided. It is very necessary in sciatic neuralgia to differentiate accurately the various confusing clinical pictures. As the sciatic is a mixed nerve and because of its great length, wide distribution, large size, and exposed position, it is likewise the subject of many neuritic manifestations. A differential diagnosis, therefore, between neuralgia and neuritis is sometimes difficult, and always important. It is questionable whether this differentiation can, or should, always be made, for there are, no doubt, pure neuralgias which develop into degenerative neuritides. If there is evidence of a degenerative process in the nerve, as shown by atrophy, muscular weakness, or changed electrical reactions, we should, for the present at least, consider the process a neuritic and not a neuralgic one.

There is a more or less characteristic attitude and gait in sciatica which should always be noted. Therapeutically, this is important, because it should suggest to the physician just what the patient's most comfortable posture is, for these postures and gaits are adaptations on the part of the skeletal and muscular systems to counteract pain, and if maintained long enough, may leave, particularly in the aged, definite structural deformities. Scoliosis, muscle rigidity, atrophy from disuse, are frequently consequences of a long-continued sciatica, particularly in the aged. Objective sensory changes are not particularly characteristic of sciatic neuralgia, but paresthesia and subjective sensory manifestations of all kinds are very frequent.

Treatment.—Before treatment is planned in any case of ischias, it should be ascertained, if possible, whether the pain is due to an organic process in the pelvis, in the hip-joint, or in lower segments of the spinal

column. Inquiry should be made particularly about accidents, sudden jolts, or jars, whether the sacro-iliac joint has been disturbed or not, and investigation as to the presence of diabetes, or other constitutional diseases, which may attack the peripheral nervous system. Tuberculosis, arthritic processes in the spine, new growths, located in the bone, or in the lower portion of the spinal cord, syphilitic meningeal infiltrations, etc., may show direct symptoms of very typical neuralgic pain, distributed, seemingly, in the region supplied by the sciatic nerve. Lesions at the sacro-iliac synchondrosis is of special moment in this connection. Various types of vasomotor neuroses, claudication, all may produce so characteristic a neuralgic pain that their presence may easily be overlooked and the therapeutic endeavor, therefore, be misdirected.

The therapy of ischias resolves itself, first of all, in the treatment, if possible, of any cause which might be active, or considered to be active, in the production of pain in the sciatic nerve, and after that, in efforts directed toward lessening the painful manifestations themselves.

At the present time in very severe forms of sciatica injections into the sciatic nerve, at or near its exit from the foramen, have obtained a certain vogue. The effectiveness of the injection treatment is difficult to measure, on account of the varying reports of success and failure. Alcohol, osmic acid, methylene blue, and salt solutions are the chief means by which this is accomplished. Alcohol, even in a small percentage, is a rather dangerous solution to use, on account of the danger of changing the neuralgic symptom-complex into a neuritic one, with its consequent paralysis, degeneration, and other sequences. The same would hold with osmic acid.

Finally, in very severe cases, where the usual measures are ineffective, splinting the whole leg in plaster-of-Paris bandages, with the immobilization limit well above the hip-joint, gives the most permanent relief from pain. In this way rest is absolutely obtained.

NEURITIDES

By the term neuritis is understood all the clinical phenomena which follow definite and demonstrable changes in the structure of the peripheral nerve tissue. Pathologically considered these changes may be due to processes largely limited to the structure of the nerve itself, the individual neuron elements, the parenchymatous form of neuritis, and changes in the perineural tissue, which indirectly affect the peripheral nerve structure. This latter is designated interstitial neuritis. Naturally the process is frequently of a mixed type, but the essential characteristic of the true parenchymatous process is a primary degeneration of the neuron constituents of the nerve structure.

Therapeutically, however, the result of such processes, whatever their degrees may be, is of importance. This result is a disturbance in the function of the muscle, dependent upon structural changes in its

substance. These changes lead not only to the loss of muscular power, but likewise to the loss of muscular substance and atrophy of the muscle itself. The dependence of the muscular structures upon the integrity of the nerve supplying it gives to the two elements, muscle and nerve, so intimate an anatomical and functional connection that the two must be viewed as a single structural and functional element and should be considered as such clinically and therapeutically. Therefore, in all neuritic processes which give a clinical expression in the form of a picture of functional disturbance there is, from the point of view of treatment, to be considered: (1) Disturbance in motility; (2) disturbance in sensation; (3) atrophy or disappearance of muscular substance, a combination of these three characteristics rendering the differentiation of neuritis from neuralgia sufficiently sharp in most cases to create two distinct types.

Unlike the neuralgias, in the neuritides there is a sufficiently definite etiological classification which can be made use of, and which particularly from the point of view of treatment may be made effective. Though here as in the neuralgias the classification is regional, yet it may at times be of considerable value. Two most important clinical divisions are made—the mononeuritic and the polyneuritic types. These differ rather in the extent of the process than in its special form.

The traumatic neuritides, in the usual acceptance of the term, will be considered in a separate section.

In the mononeuritic forms there are to be considered the occupational neuritides—those which complicate or follow joint processes, and the neuritic processes affecting isolated nerves. These latter are not at present perfectly clear. The polyneuritic types include these main divisions: Those produced by, or following, or accompanying acute infectious processes due to infectious organisms, known or unknown; those due to toxins or poisons resulting from metabolic abnormalities within the organism; and those resulting from poisons of an organic or inorganic sort, introduced from without the organism.

Occupation Neuritis.—There are various forms of occupation which, combining several factors, act upon the peripheral nerves in such a way as to produce organic changes in their structures. The constant use of muscle groups in the performance of some movements; the use of instruments, or implements, which cause continuous pressure to be exercised upon a peripheral nerve—that is, by its anatomical position exposed to mechanical forces of this sort, and sometimes the use of metallic poisons in the industrial process concerned, may cause a typical degenerative neuritis, followed by muscular atrophy. The symptoms will always bear a definite relationship to the kind of employment, and the localization will be in the muscle groups which are largely in use.

There are great variations in the clinical pictures presented, but they all have these things in common and cannot be separated from the nature of the employment which causes them.

The treatment for these forms of pressure neuritis, due to certain

occupations, is obvious. Either the occupation must be changed or the pressure modified so as there will be no injury to the exposed or susceptible nerve.

In those patients where occupation pressures operate in conjunction with mild or severe toxic factors, both deleterious agents must be dealt with. Medication here is largely useless.

Infectious Neuritis.—The existence of an infectious polyneuritis of unknown bacterial origin is at present freely admitted as a matter of clinical experience. The chances are that sooner or later, with the wider extension of our conception of some of the more common forms of infection of the nervous system, the so-called idiopathic form of polyneuritis will become considerably limited. However, the clinical picture is at present unmistakable. The infectious nature of this form of neuritis is deduced from the constitutional disturbance in the sense of the manifestations of an acute infectious disease. The prodromal period of vague general disturbance, with a slight rise of temperature, chilly sensation, and a general feeling of malaise are the introductory manifestations. These are succeeded in a brief time by the evidences of peripheral nerve involvement. The pains which at first were vaguely located in one or the other extremity now become more sharply referred to the typical peripheral distribution. There follows a very definite disturbance of the functional power of the muscles, with paresis, or complete paralysis as a final result. There is sometimes found evidence of vasomotor or trophic disturbance, in the way of edema, eruptive skin lesions, changes in the local temperature, etc. With the development of the neuritic symptoms, as a rule, the general constitutional manifestations begin to subside, and then gradually to disappear, until apart from the localized pain and the muscular insufficiency the patient seems fairly comfortable. Up to this point the similarity to an acute poliomyelitis is sufficiently striking to suggest that in the so-called infectious neuritis we may, perhaps, be always dealing with an aberrant type of this disease. Particularly does this seem likely when we consider the fact now freely admitted that there is a neuritic variety of infantile paralysis appearing under the picture of a peripheral nerve disease.

It is at this point, perhaps, when the most striking element of clinical differentiation appears, a point which makes it evident that our conception of anterior poliomyelitis must be considerably broadened before including in it the type of disease above described. Infectious neuritis shows a marked tendency to complete recovery without defect, while anterior poliomyelitis shows rather the reverse. This defect on apparent recovery creates so marked a difference that for the present, at any rate, the infectious form of polyneuritis must be freely admitted.

Treatment.—The therapeutic problem involved has to do with the treatment of the general symptoms as such; then the more direct consideration of the neuritic phenomena, as in many other forms of nervous disease. From the point of view of treatment, the most important single element is that of diagnosis, and it is particularly important to

determine as early as possible just what we have to deal with, whether a poliomyelitis or a polyneuritis. In the early period of the disease, particularly in the presence of an epidemic of infantile paralysis, such a differentiation may be difficult, and sometimes impossible. The specific diagnostic reaction of poliomyelitis is at the present time lacking; we are confronted with the necessity of basing our diagnosis chiefly upon the progressive development of symptoms, looking largely to the one important feature, the anatomical and physiological succession of symptom groupings.

Lumbar puncture may determine the question for some; careful neurological examination of sensation for others. Thus, an early loss of bony sensibility speaks for a polyneuritis rather than a poliomyelitis. Epicritic loss also indicates polyneuritis.

There is in multiple neuritis a more or less orderly succession of symptoms following the distribution of peripheral nerve trunks, or nerve terminals, and these terminal points are the chief initial foci in the development of symptoms.

The importance of an early diagnosis from the point of view of treatment naturally lies in the psychical influence which can be brought to bear upon the patient himself, for if the case is neuritis, a favorable outcome may generally be prophesied, while if the disease is poliomyelitic in character, a recovery, but with defect, is probable. Much can be made of this if absolute confidence can be felt in the diagnostic future of the case, and, furthermore, the earlier the diagnosis can be definitely made the more forcible becomes the influence of future recovery upon the patient.

The chief manifestation of polyneuritis to be considered from the point of view of direct treatment is, naturally, the pain, which in the early period of the disease may be very severe and at times almost unbearable. Our chief reliance must be upon carefully planned physiological efforts as distinct from the use of drugs. Of all these the most efficient is rest and a correct, comfortable position. Both of these, to be efficiently carried out, must be efficiently planned. No amount of rest will give relief if the position of the extremity affected is not comfortable to the patient and as far away as possible from the sensation of strain and unnatural position. That position should be selected which gives the patient a distinct sensation of relief and some approach to comfort. The extremity should be maintained in this way not only in absolute rest, but the patient himself should be taught to repress, if possible, every impulse to movement as well. It is here that the practical therapeutic application of the diagnosis comes in. Most patients can be easily led into this state of mental coöperation if the future holds no terrors in the way of a permanent invalidism, or defect for him, and if early in the course of the disease this is made clear, and if it is also insisted upon that violation of these simple rules will lead to, perhaps, permanent defects, due to bad position, then the coöperation of the patient can usually be easily obtained.

For the pain itself our chief reliance should be placed on the milder

analgesics, and in their judicious and temperate use lies much of the art of handling a patient with this disease. The salicylates in one form or another, with aspirin, or some of its modifications, are the safest drugs to use. An occasional dose of morphine may be necessary if the pain is so severe as to interfere seriously with sleep. After a time, when the acute neuritic manifestations have shown some tendency to subside, hydrotherapeutic measures should constantly be adopted. This requires no particular apparatus. Hot applications carefully placed upon the painful extremity, with the temperature as high as the patient can bear, this repeated at intervals of an hour or so during the period of most active pain, gives very definite relief. Especially efficacious is this method if the patient's painful extremity can be maintained at the same time in a comfortable position.

It is characteristic of all forms of multiple neuritis, and, indeed, of neuritis in general, that the duration of the disease is apt to be prolonged. This is due to the apparently vigorous resistance of the peripheral nerve structure to infections and poisons, consequently recovery based upon anatomical regeneration is correspondingly slow and tedious. The mistake is frequently made of miscalculating the probable time of the disease, in order to encourage the patient, consequently there is created in him, due to his disappointment, a feeling of restlessness and anxiety, based upon the fact that he has not fulfilled his physician's prophesy in respect to the duration of his illness. For this lack he blames himself, and there is suggested to him the feeling that either his disease is more serious than was at first supposed or that he does not respond to treatment, as is usually the case. In either instance, or both, there results a mental state which unnecessarily hampers the efforts of the physician and nurse.

Short of two months no prognosis in point of time should ever be given. As a matter of experience, multiple neuritis of the infectious variety extends rather beyond than short of this length of time. A word should be added here of a possible complication in multiple neuritis met with occasionally. This is the possible involvement of the vagus, or what is now called the vagal system. Such cases run a somewhat rapid pulse even after constitutional symptoms have practically disappeared. It is such patients who sometimes show evidence of complete collapse, even sudden death, for no possible reason that can be determined but that of a complete paralysis in the vagal mechanism. It is a curious fact that in asthmatics such conditions seem more likely to be found, and in this class of cases particular care should be exercised in regard to sudden movements, excessive emotional reactions and a too hasty convalescence, with a too rapid use, or attempted use, of the weakened muscles. Just what this relationship may ultimately turn out to be cannot at present be prophesied, on account of the small number of cases that have been studied.

The point where more active treatment of the paretic or paralyzed muscles should be executed can be measured by the single factor of pain on movement, whether active or passive. No effort at exercise,

massage, or electrical stimulation should be made as long as the patient experiences the slightest pain in consequence of these manipulations. When, however, the patient's extremities can be moved passively without pain, then active massage, electrical stimulation of muscle groups, passive movements, resistive movements, etc., should be carried out methodically and after a careful planning toward physiological restoration of muscle function. It must be clearly understood that the use in this instance of all these methods have practically the same aim; that is, mechanical excitation of the muscles, whether by means of electrical or passive movements, or any other form, makes little difference. There is nothing specific in the action of electricity in causing muscles, or muscle groups, to react—it is merely a convenient form of muscle innervation, in place of the normal mechanism.

At this stage of the treatment the active and helpful coöperation of the patient can be obtained by means of a careful explanation of the muscular state by assurance of the complete restoration of function, even when atrophy is very evident and apparently excessive.

During the whole course of the disease care should be taken to avoid deformity of a structural kind, by a carefully planned position of each joint. The pull of the stronger flexor muscle group should be prevented by overcorrection of the antagonistic group and by frequent manipulation, with such an end in view.

Recovery, or improvement, will first become manifest in the legs, while the return of function in the arms and hands will be slower, owing probably to the more complicated and delicate muscular interrelations. Consequently, it is advisable to make use of this fact, by planning to have the patient assume an upright position as soon as possible and to be out of bed at the earliest moment that is safe. The physical gain is a great advantage; it makes the succeeding treatment of the case much more easily carried out. If in spite of all attempts to maintain correct positions there appears a tendency to permanent shortening and joint deformities, then the extremity should be immediately encased in plaster with the joint affected in an overcorrected position. The apparatus should be so constructed as to admit of easy observation. When the correct position is finally obtained the plaster should be removed and the joint kept in the normal position by manipulative measures and by the maintenance of correct functional relationship.

There has been some discussion as to whether the faradic or the galvanic current is preferable. It is supposed by some that one or the other has a distinct specific advantage. It is probably true that neither the one nor the other exercises more than a mechanical effect, and that the one should be selected which easily and painlessly gives the desired reaction. Whether the nerve itself or the muscle be the point of stimulation are matters of complete indifference—wherever a quick reaction of muscle groups, with the least possible pain to the patient and the smallest amount of current can be obtained, there is the point of advantage for the use of the electrical current.

It must be admitted that in spite of the generally favorable out-

come of this variety of neuritis, there are cases with an apparently definite tendency not only to a very slow recovery, but to a defective one. In such cases probably the regenerative power of the peripheral nerves has become exhausted and inadequate. To know these cases in advance is apparently impossible at the present time, and there remains always the question whether they are really not to be considered as belonging to an atypical group of anterior poliomyelitis rather than to multiple neuritis.

There are a number of varieties of polyneuritis which are somewhat rare in this country—at any rate, which from a therapeutic point of view offer no particular problems unassociated with the foregoing description. The peripheral manifestations of leprosy and beriberi belong rather to a consideration of these diseases than in the consideration of the therapeutics of neuritis. Beriberi is probably an epidemic form of neuritis the exact nature and origin of which are not fully known.

Landry's paralysis occupies a position of some uncertainty at the present time. The clinical picture of an acute ascending neuritis, with grave constitutional symptoms and frequently fatal outcome, belongs to our present conception of multiple neuritis. The pathological findings of this disease are so much at variance with its clinical position as to be extraordinarily uncertain. The further the problem of anterior poliomyelitis is cleared up, however, the more reasonable it appears to include this type among the somewhat atypical manifestations of that protean disease.

Diphtheria-polyneuritis.—The most common form of the known infectious polyneuritis is that, without doubt, following, or complicating diphtheria. Its frequency is given by Rolleston as 22 per cent. in a series of 1500 cases. The neuritis following diphtheria has a very direct therapeutic interest. Ever since the period in which the serum-treatment of diphtheria was discovered and became universally used much discussion has taken place on the point of its use as a preventive, and its further use as a direct means of treatment after the neuritic symptoms have developed. Of one thing we can be certain, that as far as we at present can determine, the neuritis itself does not seem to be dependent either upon the severity of the initial infection or upon its rapidity of development, or upon its resistance to treatment. Therefore, as far as can be seen, every case of diphtheria is a possible case of neuritis. This point cannot be too emphatically insisted upon, and has its direct importance in the prevention of a too early return to activity, or convalescence, particularly in the case of a child infected with the diphtheria organism, in whom, as a result of the early administration of a sufficiently large dose of antitoxin, the local and constitutional symptoms of the disease have practically disappeared.

The period when the neuritic manifestations first appear varies from a few days to two weeks or more. As a rule, the first indication that the toxin has attacked the peripheral nerve mechanism is when the soft palate and uvula show evidence of paresis, or a complete

paralysis. As a rule, the involvement is bilateral, objectively demonstrated by the flaccidity of the soft-palate muscles, by the lower position of the uvula and, clinically, by the sudden appearance of nasal speech, regurgitation of liquids through the nose, and difficulty in deglutition. At this point the patient swallows only liquids, or semiliquid substances, and frequently cannot do that without regurgitation.

From then on the development of the neuritis varies; it may stop and apparently involve no further nerve groups, or it may develop into a fairly complete picture of multiple neuritis, with the favorite seat of localization in the extremities; or it may be strictly confined to the vago-hypoglossal-pharyngeal system. The complete and early involvement of the latter presents one of the most serious complications that we have to face in diphtheria, and one which is sometimes fatal, even in spite of the apparently successful use of antitoxin on the other symptoms.

The central fact of this symptom group is the importance of the vagal involvement, for about this is centred the chief problem of treatment, and the final outcome depends upon maintaining its integrity. The involvement of the vagus manifests itself in a variety of abnormal phenomena centred around the neurogenic activity of the heart. The muscular function of the heart itself is seldom involved, or, at most, in a secondary manner. The pulse is irregular, feeble, and intermittent. The heart is not enlarged and, as a rule, shows no evidence of valvular incapacity. The pulmonic second sound is sometimes found accentuated in spite of an apparently feeble impulse at the apex. The essential feature is the disturbance in the regulatory mechanism of the cardiac function. This is the distinguishing feature as opposed to the muscular inadequacy in many other forms of cardiac involvement following or complicating acute infectious diseases. It presents therapeutically the chiefest difficulty, for it is obvious that in the presence of a neuritis of the vagal system, we are left positively without a single therapeutic agency that has any direct effect upon the nervous mechanism of the heart. The only exception to this is absolute and complete rest. Other than this, care should be taken that no solid food is given, to avoid both the danger of deglutition, pneumonia, and sudden collapse, due to the exertion caused by choking and difficulty in swallowing.

With the further development of the neuritic process, the extremities become involved and the patient presents a picture of a rather mild form of lower extremity neuritis, with marked ataxia, weakness, absence of knee and Achilles jerks, and other evidences that are commonly met with in polyneuritis.

Apart from the vagal involvement, the diphtheria polyneuritic therapy offers no unusual problem. The almost constant and complete recovery of muscular power in the leg, with the return of normal stability and power of locomotion, suggests that beyond rest, massage, good feeding, especially of fats, and perhaps electricity, there is little that need be done. Attention might be here directed to the measures neces-

sary to prevent deformity, by maintaining a correct, or overcorrected, position of the paralyzed extremities. With the vagal situation there is needed most constant watchfulness, absolute rest, and carefully directed nursing. The child should not be permitted to move, should be fed, and every possible physical or emotional disturbance should be avoided.

Of some importance is the question of using repeated and constantly increasing doses of antitoxin in the routine treatment of polyneuritis. There is a wide divergence of opinion on this subject, and no solution seems possible at the present time. French pediatricists strongly recommend this procedure, but the evidence in its favor is scarcely convincing. Considering the almost certain favorable outcome in diphtheria-polyneuritis, with the vagal form as an exception, it would seem to be wiser to reserve this intense treatment for that type only. There are so many grave objections to its use, that, on the whole, the neuritic complication might safely be left to other less heroic measures and the diphtheria itself be regarded as the only logical object of antitoxin treatment.

Other Infectious Neuritides.—The occurrence of neuritides following various other infectious diseases is now well recognized, though in some of them, such as typhoid and tuberculosis, the causal connection is not altogether clear. It is altogether reasonable, however, to assume that if symptoms of polyneuritis develop in the course of an acute, or chronic, infectious disease, there is some connection between them and the infection. It is believed that the causative factor is the toxin of the original disease acting upon the peripheral nervous system that is rendered less resistant by the long-continued illness of the patient.

Influenzal Neuritis.—Following an attack of *influenza*, or coincident with its active manifestations, pain in the course of the peripheral nerves is common. This is ordinarily regarded as a neuralgia, chiefly on account of the absence of degenerative symptoms. The distinction between this and a true neuritis is, naturally, somewhat elusive in many instances, but an undoubted neuritis, very often of the plexus variety, does undoubtedly occur, and may in some instances be accompanied by all the evidences of a peripheral nerve degeneration, paresis of muscle groups, atrophy of muscles, and electrical changes. The distinctive feature, if there is one, is the predominance of pain over the motor symptoms.

The course of the influenza neuritis is apt to be very tedious. The atrophy of muscle groups, if found, may often be due to the non-use of muscles, on account of pain accompanying movement or attempted movement.

There is no specific treatment, and the measures that have been referred to in the treatment of the more general forms of neuritis are here to be followed out.

Typhoid Neuritis.—Neuritis in the course of *typhoid*, showing itself chiefly in the long convalescent period, is now a fairly well-recognized occurrence. It is found sometimes as a well-defined polyneuritis,

chiefly affecting the lower extremities, with all the typical symptoms of paresthesia, pain, atrophy, and degenerative changes in the muscles. The neuritis, as a rule, is rather strictly motor in type. The prevailing clinical picture is therefore a paresis, or complete paralysis, with the resulting motor incapacity. Its course is apt to be long, due probably to the general state of organic insufficiency which the long-continued typhoid is very likely to cause.

In cases of typhoid neuritis, carefully planned exercises, mild electrical stimulation, and gradual training in walking and standing are the measures that are successful in the majority of cases.

Care must be taken not to confuse the clinical picture of atrophy following the well-known occurrence of joint changes in typhoid arthritis with the neuritic manifestations. The so-called typhoid spine and other arthritic processes produce such a degree of motor insufficiency and are accompanied by pain and other evidences of an inflammatory process, that there is some chance of confusion, particularly so as the joint changes are frequently accompanied by all the evidences of a recognized neuritis. This forms part of the local process in the joints themselves; in other words, there is presented for interpretation sometimes atrophy of the muscles in the neighborhood of joints, which may depend upon two factors: A real neuritis as a part of the joint manifestations themselves, and atrophy following the long-continued disuse of muscles owing to pain or other mechanical obstacles to the use of the diseased joint.

Tuberculosis and Pneumonia.—The occurrence of a neuritic process in *tuberculosis*, *pneumonia*, and many other infectious diseases may be admitted, but therapeutically their treatment requires no special mention. Attention should be called to the severe forms of polyneuritis occasionally seen in pneumonia, or following it, with particular emphasis upon the severe grade of the infectious process and its widespread distribution. Both arms and legs are affected, being equally involved, with manifestation of pain of a strictly neuritic type.

Whether the neuritis seen in typhoid and pneumonia depends upon the bacterial agents involved in the original disease or not is, naturally, an unsettled question. There are so many other possible causative agents in diseases which produce so pronounced a metabolic change in the body that such a manifestation may be due to a great variety of causes which at present are not known.

Diabetic Neuritis.—Of the diseases depending upon metabolic anomalies (and for the present it is assumed that diabetes belongs to this class) there is none more common than that of a diabetic origin. In this disease the neuritis may be looked upon as the most common symptom. The fact that the Achilles and knee-jerks are often absent suggests that degenerative changes in the peripheral nerves of a neuritic character might be the causative factor. The neuritis is probably purely toxic in origin and bears a close relationship in this respect to that found in the alcoholic. It is almost always motor in form and is chiefly found in the lower extremities. There is seldom a

very frank manifestation of a widespread neuritic involvement to be found. In most cases the absence, or marked diminution of the deep reflexes of the legs, with some tendency to weakness of the muscles, lowered faradic reaction, and paresthesia are the chief definite symptoms to be found. It must also be remembered, however, that at times a neuritis with a marked sensory reaction is to be found, an acute pain of a very intense character, typically neuralgic in type. The sensory type is chiefly mononeuritic in distribution.

Treatment.—Apart from the general therapeutic principles that have already been laid down, there remains a treatment directed to the positive agent; that is, the diabetes itself. It is a rather common experience to see the sensory manifestations of a diabetic neuritis grow less or disappear, coincident with the lessening of the sugar percentage. This, however, does not appear to have any effect upon the motor symptoms at all, and the reflexes do not reappear, in spite of the total, or almost total, absence of sugar in the urine.

The common occurrence of a rather marked degree of arteriosclerosis in diabetes, as well as secondary nephritis, should preclude the use of any very intensive physical form of therapy. Naturally, in the neuritis found in cases of diabetes great care should be taken not to use methods which might injure the skin. Pressure, or mechanical measures of this kind, should be used only with the greatest care. Therefore, the use of splinting, adhesive bandages, or plaster-of-Paris casts, cannot be recommended as routine treatment. Rest is, perhaps, the best therapeutic agency that we have, and this, together with the general treatment of the patient, is about all that can be used with any degree of safety.

Toxic Neuritides.—The neuritic manifestations produced by poisonous material introduced from without the body are of great importance. They are particularly to be recognized, as they form an important chapter in industrial diseases; that is, diseases which are produced by the use of poisonous material in the arts, such as lead, arsenic, antimony, bismuth, etc.

Alcoholic Neuritis.—In addition to the occupational form of toxically produced neuritis, the most important is that following the use of *alcohol*, on account of its frequency and likewise on account of its more or less typical clinical characteristics. However common, its frequency cannot be easily determined, but it is certainly by far the most frequently seen in a hospital service where alcoholics are freely admitted to the wards. Many attempts have been made to bring into relationship with the occurrence of alcoholic polyneuritis the manner and the amount, as well as the nature of the alcoholic drink which is ingested. Whether the chronic drinker alone is subject to this disease, or the occasional drinker, and the amount of alcohol taken is in excess of his average, or when the alcohol is more concentrated as to form, or whether there are other unknown factors, are still questions in debate.

A rather frequent occurrence of polyneuritis as incident to, or following, an attack of delirium tremens has often been noted, and would

suggest that other factors are at work in the production of the neuritic manifestations that are at present unknown. There is likewise a tendency for alcohol to attack the peripheral nerves, which, on account of occupational overuse are apparently less able to resist its toxic influence.

In addition, unnatural positions of the extremities, which put undue strain on the plexus in positions maintained for long periods of time, render them more likely to be attacked by a neuritic process than would otherwise be the case.

Alcoholic polyneuritis presents a very typical picture, with certain prodromal signs, which in an individual with excessive alcoholic habits should always arouse suspicion. The occurrence of paresthesia in the legs of a very annoying character is frequently the first symptomatic manifestation. Sometimes the process apparently only goes as far as this, and the definite neuritic manifestations stop at this point. In the typical cases, however, this prodromal period of paresthesia is followed by a gradually increasing motor weakness, with the characteristic extensor localization. In addition to this, there is developed exquisite tenderness along the course of the nerves. So severe may this become, that the slightest pressure, even of the bed-clothes, for example, cannot be borne. The occurrence of a quickly developing and high degree of atrophy, together with marked weakness, paresis, or paralysis of the extensor group, particularly in the legs, finally establishes the completed picture. The muscles show the reaction of degeneration and a lowered faradic excitability. The legs are much more involved than the upper extremities, and there the characteristic wrist-drop, due to extensor involvement, gives a definite appearance of a neuritic process that is very widely spread.

The course is apt to be very long, the minimum duration two months, and recovery frequently is not complete for a year or more.

TREATMENT.—Complete withdrawal of all alcohol is the first step in the treatment. Then prompt elimination by baths, catharsis, and diuresis. Then overfeeding particularly in foods rich in digestible fat. Milk is of primary importance. An extremely important feature is to prevent deformity due to stretching of the tendons of the extensor muscle groups. Owing to the high degree of muscular weakness, the acuteness of onset and the great degree of pain, the antagonistic pull of the lesser involved groups of muscles is not resisted by the patient, he being intent chiefly upon maintaining positions which give him the maximum degree of comfort. Consequently, faulty positions are very easily produced, contractures and deformities naturally following. The first consideration, therefore, must be directed to the maintenance of correct or even overcorrected positions from the very beginning. Nothing at the initial stage is so effective as sandbags, which give support to the extremity in a correct position. The use of plaster-of-Paris bandages encasing the leg would be of great advantage if it were possible to employ them, but on account of the intense sensitiveness of the skin overlying the muscles, this is seldom prac-

ticable. The splinting of the extremity needs constant attention, for on account of pain, mental excitement, restlessness, and other manifestations of the alcoholic temperament, such patients are difficult to control and manage.

Splints should be easily removable so as to be able to practise forced extension. Patients with alcoholic neuritic contractures do well under such manipulations.

For the pain itself, recourse at times must be had to morphine, for rest is essential at first, in order to prevent complications. It is only after the pain has subsided that other measures are in order. So that, as a rule, the stage of active treatment must be instituted rather late, long after the degenerative process of the muscles has set in.

The therapeutic difficulties of a case of alcoholic neuritis are not confined to the treatment of the nerves and muscles. Above all, the individual should be considered. The alcoholic subject offers problems in nutrition, sleep, and mental states of a very difficult nature. The withdrawal of the alcohol, probably of prime necessity for the complete recovery of the patient, as far as his neuritic symptoms go, needs careful consideration.

The danger in delirium tremens of an ever-present possibility of the group of symptoms associated under the term alcoholic edemas is an additional element in the therapeutic problem presented by these cases. In threatened alcoholic edema, wet brain, washing of the stomach is of great service, in addition to the eliminative treatment suggested. Paraldehyde is a useful hypnotic to replace the alcohol.

PROGNOSIS.—The prognosis, as a rule, is good, but complete restoration depends upon the care with which the measures are instituted to control, or to counteract, the dangers which have been alluded to. No favorable unconditioned prognosis should ever be given until the period during which complications might develop is long past. Electricity, massage, and other mechanical forms of treatment should only be begun when the sensitiveness of the nerves and muscles has completely disappeared, and all such measures should be begun very gently and increased very gradually. As soon as the patient can bear the weight of the body, he should be encouraged to attempt to stand and walk and get out of bed. Passive movements should be instituted at the earliest possible moment, and should be encouraged as a part of the routine treatment even after the patient is able to move about.

Lead Neuritis.—*Lead* neuritis is one of the common forms of occupational diseases, and is one that is used most frequently to illustrate the dangers of certain occupations. It is found most often in workers whose trade brings them into relation with lead in some of its various forms. It is found most frequently, therefore, in lead-workers in reduction plants in which lead forms a part of the chemical process, in the making of articles in which lead is an important part of the product, and in various other kinds of metallic work.

It does not concern us in this place to discuss the means by which

lead, or its products, is conveyed into the body, whether by inhalation, or direct contact. These are matters which lie apart from the present subject. One point should, however, be here insisted upon; that is, that in the presence of any characteristic peripheral neuritis of unknown origin, care must be taken to investigate the possibility of lead being an important factor. Lead is used in a great many trades which at first sight would not arouse suspicion of its use at the hands of the clinical investigator. It is therefore advisable to consider lead always as a possible cause until it can be definitely excluded. The characteristic symptom of lead neuritis is its motor character and its practically painless course. It usually affects the motor groups of the fingers and forearm, and sometimes the upper arm, showing a marked predilection for the extensor group of the wrist and fingers, not involving, as a rule, the supinator longus group. It has been suggested, and apparently with good reason, that the muscle groups affected are those which have been made non-resistant by constant overuse, and that the specific selective quality of lead on these groups of nerves is not a factor. It is a matter of observation that in certain types of lead palsy the clinical picture as commonly presented has been much changed, on account of the different employment of the individual, groups of muscles being affected which are the subject of constant use, to the disadvantage of such muscle groups which have not been overused.

The atrophy in lead neuritis is very marked, develops quickly, and is unaccompanied by sensory manifestations. There is seldom the marked paresthesias that are found in alcoholic neuritis. It presents the characteristic clinical picture of involvement of the upper extremities, with practical freedom of the lower extremities, absence of severe pain, the presence of marked atrophy and muscular weakness, and the exclusion, as a rule, of the supinator longus.

It must not be forgotten that there are various atypical forms of lead palsy, some of which closely resemble alcoholic neuritis, and some of which are complicated by the involvement of the cranial nerves.

TREATMENT.—In consideration of the etiology, the treatment of lead palsy resolves itself chiefly into methods aimed at the withdrawal of the exciting cause. In other words, the occupation involving the use of lead must be given up, and given up completely, in the presence of a case of lead poisoning. It is further necessary to insist that that individual who once has had symptoms of lead neuritis seems to have a lessened resistance to the effects of lead on his peripheral nervous system, and, therefore, if possible, such an individual should never be permitted to work again in trades in which the use of lead forms an important element. Briefly then, the best possible treatment for cases of lead palsy consists in preventing the individual from ever again coming in contact with lead in sufficient quantities to favor its introduction into his body.

Concerning measures of a prophylactic nature for the prevention of lead poisoning in workers in lead not much can be said in this

place. Cleanliness, the wearing of gloves, inhalation masks, protection of food from contact with lead, thorough hygienic surroundings will probably in most cases prevent its occurrence.

Patients with lead palsy very often show constitutional effects of lead poisoning: anemia, with arteriosclerosis, gastric symptoms, nephritis, etc. Very often complete rest in bed for some weeks with measures planned to correct the constitutional symptoms which are present are most successful. Almost any kind of treatment which includes absence from the possibility of further lead poisoning and rest for the part affected will be efficient. It should be remembered, however, that after complete apparent recovery the muscles and nerves affected have retained a certain susceptibility to a recurrence, even under conditions which bear no relation to those under which the original disease was contracted. It is therefore better, if possible, to plan that kind of employment which will not involve the overactivity of the muscle groups which were originally involved.

The use of drugs in lead poisoning is not necessary if the measures outlined above are carried out. The iodides are traditionally recommended, but the logic of their use is not evident. They certainly cannot be regarded as having any specific effect upon the lead remaining in the system. However, as a tonic, or alterative, there does not seem to be any serious objection to their use.

Attention should be called to the form of lead intoxication which gives the picture of an acute maniacal disturbance. This is a well-recognized clinical picture. The point to be made here is that in the presence of a group of mental symptoms in a lead-worker the occurrence of a peripheral neuritis should be looked for, and if found, probably furnishes the explanation for the mental symptoms which are present.

Arsenical Neuritis.—*Arsenic* is, perhaps, the third most common cause of neuritis due to metallic poisons and as an occupational factor may be of some importance. Its use in industries has been of late much restricted, therefore its occurrence at the present time is a matter of considerable rarity. Arsenic produces a rather typical form of polyneuritis, without any characteristic manifestations, with the exception of gastro-intestinal complications of a somewhat severe kind.

Therapeutically, outside of the withdrawal of the source, the measures outlined above will amply suffice.

Various other metallic poisons used in the arts produce at times neuritic manifestations, as well as drugs, which may have an apparent specific affinity with the nervous system, veronal, trional, etc.

Therapeutically, the disuse of the drugs or the withdrawal of the offending sedative is all that is necessary. Care should be exercised to avoid the confusion of the accidental presence of a suspected etiological agent with the occurrence of the neuritis, or to be necessarily impressed if the withdrawal of the agent causes the neuritis to disappear. The presence of a localized neuritis as a coincidental occurrence

should always be remembered. The occurrence of neuritis in chronic morphine-users or other constant users of drugs in which the hypodermic needle is the agent for its introduction into the system should be noted here. The question is undecided as to whether the neuritis is the effect of the constant trauma produced by the introduction of the needle or is due to the drug itself. These cases are so rare that they are only mentioned in this place as a matter of completeness.

Neuritic Manifestations in Tabes.—The occurrence of neuritic manifestations in tabes dorsalis should be noted here for the sake of completeness. It has been observed for a long time that in certain cases of tabes dorsalis there was marked evidence of muscular atrophy, which could not be explained by disuse, or by the joint changes, or any of the manifestations of tabes associated with muscular insufficiency. It was assumed that, in addition to the purely sensory features of the tabetic process, there was a degenerative process in the motor system, and that this degenerative process had probably its origin in the peripheral nerves, somewhere in their course. It will not be necessary here to enter into a theoretical discussion as to whether this process takes place in the motor roots of the spinal cord or not. Therapeutically there is naturally very little to be done, as the degenerative quality of the neuritic manifestations is very evident, and probably depends upon the same kind of process that is located chiefly in the sensory neurones.

There have been observed and reported from time to time cases of peripheral neuritis which seemed to be based upon a syphilitic infection; a number of instances are found in the literature. The peripheral neuritic manifestations of syphilis do not differ in any essential way from that of other infectious processes. The existence of polyneuritis dependent upon syphilis is very questionable at the present time, although its presence as a part of the syphilitic manifestations should be considered. Naturally, the chief therapeutic indication, outside of the general rules of treatment laid down in the treatment of neuritides elsewhere, would be the treatment for the infective agent.

There has of late been much interest aroused in the consideration of neuritis depending chiefly upon arteriosclerotic processes, the so-called *arteriosclerotic neuritis*. There have been reported thirty or forty cases in the literature which seemed to depend largely upon the presence of arteriosclerosis of a more or less generalized kind.

The distribution of this form of neuritis is rather uncommon. It was found chiefly in the distribution of the sciatic nerve. In some of these cases a disturbance of the bladder mechanism was also present. The chief diagnostic feature of these cases is the pain along the course of the nerves without the presence of sensitive points of pressure.

The relationship of this form of neuritis to intermittent claudication should be remembered.

As far as treatment is concerned, there is no direct treatment for a neuritis of this sort, and efforts should be made to treat the general condition, that of arteriosclerosis.

Mononeuritis.—The occurrence of inflammatory processes in single nerves, or plexus, is very common, and they offer for examination the essential symptomatic manifestations of neuritic processes elsewhere. The dividing line between them and neuralgias is sometimes very tenuous. We are forced to regard the presence of paralysis and muscular insufficiency as an important element in the differentiation and diagnosis. In some of the mononeuritides there seems to be an undue selective action on the part of the causative agent. This apparently lies either in a special quality of this agent or a lessened resistive power of certain nerves to the action of this agent. This may be due to certain anatomical defects, which place the exposed nerve in a more favorable position for the action of the agent, whether it is toxic or mechanical. The overuse of muscle groups in trades, or in athletic exercises, or even in the normal movements, sometimes undoubtedly causes such a lessened resistance, and the motor-nerve mechanism is thus more easily attacked by processes, whatever their nature may be, which have as an end result inflammatory conditions of the nerves. The cranial nerves may be subject to such isolated neuritic processes, either in their central or in their peripheral courses, and it is of some importance therapeutically that the latter differentiation should be made. The diagnostic importance lies in the fact that the prognosis, as a rule, in the peripheral variety is generally to be regarded as favorable, while the reverse may be said to be true in the central variety. The judicious use of a good prognosis in a therapeutic way has been previously alluded to. The cranial nerves, singly or in groups, may be the subject of neuritic processes as a part of a more general condition, similar to multiple neuritis, or singly as a complete cranial phenomenon. As a rule, when more than one cranial nerve is affected, the original factor must be looked for outside of the cranial cavity, except in cases where purely mechanical factors may be assumed to exist, as in brain tumors, meningitis, traumatic agencies, and various sorts of things which act in a purely mechanical way. The contiguity of the process, its extension by virtue of anatomical propinquity, forms the essential pathogenic consideration. The extension of the inflammatory process to the nerves lying in its immediate vicinity is sufficiently obvious to give a key to the solution and a sufficient basis for treatment directed to the cause of the inflammation.

The subject of central neuritis is so vague at the present time to preclude any direct therapeutic importance, and belongs rather more to the special subjects concerned.

Third Nerve.—The third nerve is especially liable to neuritic processes, on account of its long course and its exposed position. In the presence of ocular manifestations pointing to its involvement the first consideration is in the interpretation of the cause, and following that, the proper treatment. Obviously, there can be no direct treatment of such a condition in the approved manner as already laid down. In regard to the third nerve, there is a somewhat rare form of neuritis which includes in its clinical manifestations ptosis, narrowing of the

palpebral fissure, and a reactionless pathologically small pupil. This points, naturally, to a direct involvement of the sympathetic in the neck, and is apparently found dissociated from other manifestations of a general neuritic character.

In those cases in which a tumor, exostosis, or other mechanical factors can be excluded, there remains for treatment these abnormal manifestations, forming a more or less complete picture. It is found that such conditions clear up rather rapidly under strychnine and electrical stimulation of the levator muscles.

Fifth Nerve.—The involvement of the motor branch of the fifth as a mononeuritis is practically unknown, but if together with a typical quintus neuralgia the motor branch is found to be involved, then we have to consider the probability of a neuritis being present. This is always the case when in addition to pain there are concomitant motor symptoms. In the presence of this combination, the probability of a mechanical cause should immediately direct the attention toward the place where the motor branch most closely approximates the sensory distribution of the fifth; that is, the *cavum Meckel*. The usual etiology that has been found in the rare instances of such a combination is a tumor that is located there, or in the immediate neighborhood.

Tumors of the Gasserian ganglion have been reported in enough instances now to establish a somewhat typical clinical picture. Naturally, the therapeutic indications are those which the presence of a tumor in this region should suggest and require no further mention here.

The question of a typical trigeminal neuritis as opposed to neuralgia has previously been touched upon, and the therapeutic problem has been often pointed out as solved by the diagnosis. In this connection it might be noted that the possibility of a purely syphilitic neuritis of the total distribution of the fifth should always be thought of, and in the absence of any other explanation, the use of the proper specific treatment, either mercury or salvarsan, should be carefully considered.

It can be seen from this brief description that the interpretation of symptoms in the distribution of the fifth may be a matter of great confusion, and the therapeutic indications would, naturally, share this difficulty of interpretation. It should be remembered that it is not essential whether we regard a process in its distribution as a neuritis or as a neuralgia, because if relief is to be obtained, it depends largely upon the etiological factors. It is obviously impossible in this connection, as it is in others, to determine absolutely how much of the process should be considered neuralgic and how much neuritic. It is likewise true that the therapy, if it is to be successful, would be as efficacious in the neuritic as in the neuralgic processes.

Sixth Nerve.—The sixth nerve can undoubtedly be affected by an isolated neuritic process, usually as a symptomatic expression of a more general process, which is as yet either quiescent or objectively not clearly manifested. It is an early indication in tabes, paresis, and general syphilitic infection of the nervous system. Its paralysis must be laid down as a pure neuritis. The predilection of this nerve to

syphilitic toxins is due, no doubt, either to its structure or to its anatomical position. Its occurrence in diabetes and other metabolic anomalies is less certain as well as its occasional appearance in other states dependent upon diphtheria and, at times, in chorea. Its obvious treatment depends solely upon the causes which are back of its clinical manifestations. In the presence of an apparently isolated neuritis of the sixth nerve the greatest care must be taken to exclude constitutional states, of which this paralysis is only a symptom. It is very questionable whether the sixth nerve alone can be the subject of a neuritic process without other symptoms being present to indicate its source. It is easily seen, therefore, that therapeutically a neuritis of the sixth depends upon the therapy applied to the general condition, of which it is one expression.

Seventh Nerve.—The seventh nerve, on the contrary, is the most common cranial nerve by far which is the subject of a neuritis. Its wide distribution, the multiplicity of its functions and its curiously exposed anatomical position make it an easily reached focus for attacks of every kind of inflammatory processes. It is not only inclosed in the temporal bone for quite a part of its course, but the channel through which it goes is very tortuous. It is very close to the most common source of bone infections in the cranial cavity; that is, the middle-ear processes. Its most common variety of reaction to infections is the complete paralysis of its peripheral distribution, the so-called Bell's palsy, in more modern terms to be known as a Fallopian neuritis, meaning by that that the initial neuritic process takes place somewhere in the Fallopian canal. The cause, or causes, are somewhat a matter of conjecture at present. That a typical neuritis exists is amply proved by microscopic studies, which have shown that a neuritis in that portion of the nerve inclosed in the Fallopian canal can be demonstrated. This neuritis differs in no way histologically from that of a peripheral nerve subject to trauma or intoxication.

The distinction of whether we are dealing with a process in the canal, or peripheral from it, or central to it, is from the standpoint of treatment not important. The real therapeutic question, however, is whether we are dealing with a neuritis of the peripheral branches of the seventh, or one that has its origin and seat of location in the central portion of the seventh nerve. The distinction is sufficiently obvious if the anatomical problem is taken into consideration. Any process of a neuritic character affecting the seventh in any part of its peripheral distribution must invariably produce a paralysis of all its branches. As a matter of experience, leaving the hypothesis as to causation in abeyance, a neuritis, or neuritic-like process attacking the seventh in its centre, leaves untouched its upper branch. In addition, naturally, the process as an isolated central affair is obviously almost an impossibility, and so the distinction may readily rest upon this one fact, attested by repeated clinical experience. It is important, naturally, to keep in mind the rare exceptions, and when face to face with them, the proper recognition can readily be obtained. The importance of the

diagnostic differentiation is at once apparent from two points of view, at least: The comfort that the assurance of a practically constant ratio of recovery, in the peripheral variety, to the patient, cannot be overemphasized; and, again, it is mainly this form that lends itself to treatment directed locally to the paralyzed nerve; that is, the paralysis of the central mechanism of the seventh being a part of a more composite clinical manifestation, is, naturally, not open to direct therapeutic treatment. The complete paralysis of the seventh produces a curious sense of discomfort to the patient apart from the loss of muscle control itself, the clumsiness in speaking and eating, the sense of awkwardness and awareness, to say nothing of the occasional somewhat severe pain over the mastoid region, offer very definite points of therapeutic endeavor.

The duration of a typical facial paralysis can be considerably shortened by active electrical faradization on the chief motor points. This should be begun only after the paralysis has existed for a few days and after the pain has practically disappeared. Faradization, or galvanization, should be at all times as gentle as is consistent with obtaining slight functional response. It is evident that the only virtue of electrical treatment in such cases lies in the ready means which it offers for a method of mechanical stimulation. There is no other way known to us of producing movement in the muscles supplied by paralyzed nerves but this. It is probably true that the muscles are thus brought into a more normal relationship with their blood supply following movement, no matter how produced.

It seems reasonable to conclude, therefore, that contractures resulting from the unopposed activity of the unaffected antagonist group will be counteracted, and the most inopportune result that disturbs the usual course of a Bell's palsy will then be avoided.

Gentle massage and the gradual use of exercises as soon as voluntary power has shown its initial return are the therapeutic means that are required. No medication is necessary, except in cases in which there is considerable pain. The time-honored use of strychnine can be discarded, as in small doses it has only a slightly tonic effect on the general system, and its use in very powerful doses is absurd, because to attempt to stimulate a completely paralyzed group of muscles dependent upon a neuritic group process in its supplying nerve is the height of therapeutic stupidity. The patients, as a rule, need no other treatment, with the exception of the mental stimulation which a favorable outcome of such an affliction guarantees. This can be almost always given to them. Naturally, there are some cases which do not recover completely, and which leave as a result some facial asymmetry, due to the lack of permanent regeneration of a branch of the facial distribution. There is no particular way by which, in a given case, this lack of complete return can be foretold, but it is well in the beginning of a typical case of facial paralysis of the peripheral type to warn the patient that there is a possibility that an absolute return to perfect facial symmetry may not be obtained, but that the chance for this to happen is very small.

In the cases of facial paralysis produced by trauma, or the results of operation on the mastoid process, by which the facial is cut in the course of the operation, the result, naturally, is not to be considered as favorable. Such cases, however, do not fall within the province of this article.

The occurrence of a bilateral facial neuritis, a double Bell's palsy, is occasionally met with. Clinically, its recognition is easy if the possibility of its occurrence is remembered. The mask-like appearance of the face and total inability of the patient to give expression to emotional feelings and the complete paralysis of both facial territories, with the wide-open eyes, are sufficiently characteristic. Unlike the unilateral variety, the bilateral form cannot be regarded as a neuritis depending upon a localized developing process, such as is found in the Fallopian neuritic type, but must be regarded as a form of polyneuritis, with a localized mononeuritic character.

The most common cause of neuritis of the facial bilateral in distribution is due to general infectious processes, which, for some reason or other not wholly understood at present, become localized in the facial distribution. Syphilis is supposed to be a possible cause but only in rare instances.

A form of bilateral facial palsy met with occasionally is that resulting from basal fractures, in which both facial nerves are caught before they emerge from the skull, either in the hemorrhage or in the actual destruction of bone structure. In such cases the regeneration of the nerves depends upon the extent of the original injury. What has been said of treatment applies to this form likewise, merely that the problem is rather more complicated. We are dealing frequently with an absolutely unknown etiological factor, the distribution of which in the organism is sufficiently restricted to conform to a very limited expression in both facial territories. If, however, we are able to obtain a response to faradism in the facial, it is reasonable to conclude that a return to normal is possible, and what has been said of the prognosis of the ordinary Bell's palsy can be said here, with such limitations as have been suggested of the double. The prognosis, therefore, can be said to be favorable, but the time in which complete restoration takes place is much more protracted than in the unilateral forms.

Cochlear Nerve.—A neuritic process of the cochlear nerve belongs, perhaps, to a more specialized territory than can be treated of here. That it exists as apart from generalized processes, such as syphilis, tabes, etc., and apart from a purely local condition is, perhaps, questionable. It is important, however, to know that it is found at times with Fallopian neuritis and as a part of the symptom-grouping in tumors of the brain, particularly in the pontine angle variety of the neuromas and other widespread processes involving the central nervous system. Nerve deafness means always somewhere in its course a neuritic or a post-neuritic degeneration of the fibers of the cochlear nerve. What was said of the vagus under the heading of neuralgias might be repeated here as applying to auditory neuritis. Isolated in a clinical way, with

the possibility of accurate recognition, it probably does not at present permit of a place in any therapeutic consideration of the neuritides.

Spinal Accessory Nerve.—The occurrence of isolated *spinal accessory* neuritis is a matter of great rarity, though instances are found reported in the literature. It is generally a form of pressure neuritis, caused by trauma acting directly upon the peripheral course of the nerve. The therapy presents no unusual problem that would separate it from the more general consideration of other forms of the neuritides.

Hypoglossus Nerve.—The *hypoglossus*, on the other hand, is more frequently found the subject of neuritic processes, generally, however, as a part of a more generalized condition, in which other cranial nerves are found likewise involved. As isolated, it is generally unilateral in type, and for its occurrence there is generally a local cause to be discovered, frequently of a traumatic origin.

The neuritic degeneration of both hypoglossi as a part of the progressive degeneration of the bulbar type palsy can easily be recognized, and its treatment is involved with that of the process in general.

Cranial Nerve.—In a cranial nerve involvement the distinction should be made, if possible, between the nuclear type and the purely peripheral form. The fact that the nuclear types represents very unfavorable subjects for treatment is of therapeutic importance. The reverse is true of the peripheral types, particularly those which have a possible relationship with toxic processes, or are due to the presence of poisons in the circulation, however introduced, and which may show a predilection for peripheral localization. It is fair to assume the possibility of any of the toxic, or poison-produced neuritides, and for this reason every precaution should be taken to exclude these in the search for causative agents. It must not be forgotten that clinically there may be few differences between a degeneration which is nuclear and one which is peripheral in type. Their distinction is largely based upon causes, and effort should be put forth in this direction.

Plexus Neuritides.—Plexus neuritides can be considered as a form of mononeuritis, in which the general picture becomes limited to some particular plexus, or nerve group, having more or less a common function. The distinction between the plexus and the polyneuritic form is largely clinical; that is, the same contributing cause will produce a very different end-result. For some reason not entirely clear, the plexus type shows no particular tendency to spread beyond the initial point of localization, and, further, shows no particular selection and no unusual prodroma. In other words, we have to do with more or less local causes, whatever its particular location, dependent, probably, on physiological reasons, among which a previous fatigue, or overaction, or lessened resistance, are the principal features of importance.

Leaving aside the effect of traumas, violent injuries, etc., we have to face a well-defined inflammatory process limited to the brachial plexus, and practically the same condition may be present in the lumbar, or any other grouping of peripheral nerves having a peripheral distribution.

The same distinction is to be made between the plexus and root neuritides that was touched upon in the neuralgias, and for the same important therapeutic reasons.

Treatment, apart from the relief of pain and discomfort, has largely become a question of the diagnosis, and the discovery of the anatomical distribution of the actuating factors.

The prognosis is so dependent upon the nature and localization of the causative agency that their demarcation becomes of some importance. Any process located in the spinal canal, or its osseous casing, or the spinal cord itself, may conceivably act in the production of a root process, and by its further extension into a plexus, or peripheral localization. In this way the clinical phenomena may be somewhat confusing.

The bilateral character, as a rule, belongs to the root processes, as opposed to the unilateral character of the plexus type. This differentiation is of some value in differentiating the true conditions. More and more does the question of diagnosis come into the problem, and many a failure in treatment is to be explained by the inability to interpret properly the clinical data which are obtained. Tumors of the cord, tuberculosis, osseous processes of various sorts acting mechanically, syphilis, or other constitutional states are some of the common causes of plexus or root neuritis. These conditions all would give about the same clinical appearance, and none of them can be properly or successfully treated unless the causative agent is discovered. Frequently this discovery is only made after the degenerative processes have gone so far as to preclude the normal resumption of function.

The direct therapeutic problem in plexus or root neuritis is much the same as in other forms of mononeuritis. With the discovery of the cause, our chief efforts at relief may take a direct line of action, aiming at the removal, or the modification, of the offending process, which, in the absence of this knowledge, makes our efforts generally halting and indirect. To treat merely the symptoms of pain and muscular insufficiency is to lose the chance of permanently benefiting the patient, instead of temporarily relieving him. The brachial neuritis, for example, may be but a part of the process involving the shoulder-joint, a cervical rib, a pachymeningitis, or an old injury long lost sight of. The x-ray may reveal the true actuating cause, and our main therapeutic endeavor in such instances should be directed to the shoulder, and, secondarily, to the peripheral components.

A radicular neuritis, due to pressure from meningitic infiltration, or a cord tumor, cannot be treated by massage and electricity with any chance for relief, when the more extended factor lies untouched and far removed from ordinary therapeutic efforts.

CHAPTER II

THE TREATMENT OF INJURIES TO THE PERIPHERAL NERVES

By JAMES SHERREN, F.R.C.S., ENG.

INJURY may affect a peripheral nerve either in its course (in continuity) or at its termination, one of its branches of distribution being involved. The effect of injury in these two situations must be separately considered, as the symptoms produced and the method of treatment differ.

Injury in Continuity.—This may result from penetrating wounds of accidental or operative origin, from overstretching (traction) or as the result of pressure, suddenly applied as by a blow, long continued the result of involvement in callus or fibrous tissue. The median, ulnar, and radial nerves at the wrist are particularly liable to suffer from accidental wounds; the spinal accessory, facial, last dorsal, and ilio-hypogastric are the nerves most often wounded during the course of operations.

The brachial plexus above the clavicle and the median nerve at the wrist are those most often affected by traction. The nerves of the arm from pressure, sudden or long continued.

Many nerve injuries are preventable. Such are the injuries of the last dorsal, ilio-hypogastric and ilio-inguinal nerves in kidney, appendix, and hernia operations. The symptoms produced by these injuries may be more irritating to the patient than the condition for which the operation was performed. Again, injuries to the facial and spinal accessory nerves in operations upon the mastoid process and neck are rarely unavoidable. Sufficient thought is not, as a rule, given to the care necessary to avoid injury to important nerves during the course of operations. J. P. Huguot has recently redirected attention to nerve injury during the course of abdominal operations.

Many traction injuries would not arise if the possibility of their occurrence in certain procedures were remembered. The post-anesthetic brachial palsies result from overstretching, and can be avoided by having the patient's upper limbs closely applied to the side during the whole course of the operation. Brachial birth paralysis could often be prevented if the accoucheur remembered it was due in practically every case to "pulling."

Again, injuries the result of pressure not infrequently originate during the course of surgical manipulations, for example, injury to the nerves of the upper limb from the application of an Esmarch bandage

or tourniquet, or associated with Volkmann's ischemic contracture from the pressure of splints, strapping or effused blood; to the ulnar nerve from the pressure of strapping used in putting up a fracture of the clavicle by Sayre's method; to the external popliteal from the pressure of a Clover's crutch; the musculospiral from crutch pressure, or the plexus below the clavicle from the reduction of a dislocation of the humerus by the "heel in axilla" method.

However caused, nerve injuries fall into two groups—complete and incomplete division, the word division being used in connection with the conducting portion of the nerve. Complete division with degeneration of the peripheral end may result from an injury which leaves the naked eye continuity of the nerve intact; this form of division is called physiological and is of the utmost importance from the point of view of recovery (*vide* p. 59). When anatomical solution of continuity is produced, the division is called anatomical.

The complete classification is into:

Complete division	{ Anatomical.
	{ Physiological.
Incomplete division	{ Anatomical.
	{ Physiological.

DIVISION OF PERIPHERAL NERVES

Symptoms following Complete Division.—Sensory Symptoms.—Complete division of a mixed nerve results in loss of those forms of sensibility which it "exclusively" supplies, together with paralysis of the muscles to which it sends motor fibers.

The following conclusions were arrived at as the result of the investigations of Henry Head and the writer.

The afferent fibers in peripheral nerves can be divided into three systems:

1. Those which subserve deep sensibility and conduct the impulses produced by pressure. The fibers of this system run mainly with motor nerves and are not destroyed by division of all the sensory nerves to the skin.

In a part innervated only by this system gradual increase in pressure can be recognized and pain experienced when such pressure becomes excessive. The patient can also appreciate the extent and direction of movements produced passively in all the joints within the affected area.

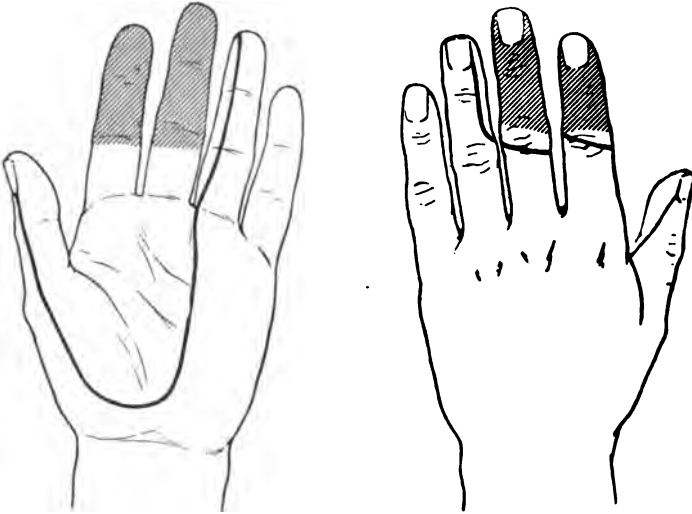
2. Those which subserve protopathic sensibility. This system of fibers and end organs respond to painful cutaneous stimuli and to the extremes of heat and cold; it also endows the hairs with the power of reacting to painful stimuli. The distribution of the protopathic fibers usually overlaps greatly the area supplied by similar fibers from adjacent nerves.

3. Those which subserve epicritic sensibility. The nerve fibers and end organs of this system endow the part with the power of responding

to light touch with a well-localized sensation. The existence of this system enables us to discriminate two points and to appreciate the difference between cool and warm. The distribution of these fibers in large peripheral nerves, such as the median and ulnar, has very little overlap compared with the great overlapping of the protopathic supply.

These investigations were carried farther, particularly with regard to deep sensibility and the distribution of heat and cold spots by Head and Rivers after voluntary section of the radial and external cutaneous nerves in the former's arm.

FIG. 1



To illustrate the changes in sensibility met with after complete division of a peripheral nerve. The area inclosed by a line is that in which epicritic sensibility is lost. The shaded area is that of loss of epicritic and protopathic sensation. The unshaded portion is the "intermediate zone."

To illustrate these changes in sensibility after division of a mixed nerve the median is an excellent example (see Fig. 1). After complete division of this nerve at the wrist, if no tendons have been divided at the same time, the patient is able to appreciate those stimuli commonly called tactile. A touch with anything which deforms the skin may be readily appreciated and correctly localized. When pricked with a pin the patient knows that he has been touched but fails to perceive the sharpness of the stimulus (deep sensibility). But if tendons are divided at the same time or the section involves the nerve above the point at which its muscular branches are given off, deep touch may be unperceived. These characteristics are of the utmost importance; many cases of nerve injury have been overlooked from failure to recognize these facts.

The point of a pin and all temperatures are unappreciated within

an area which varies somewhat in each case (loss of protopathic sensibility). Surrounding this area and corresponding closely to the distribution of the nerve as figured in anatomical text-books is a territory within which the patient is unable to appreciate light touches with cotton-wool and temperatures between about 22° and 40° C. (minor degrees of temperature), and fails to discriminate the points of a pair of compasses when separated to many times the distance necessary over the corresponding portion of the sound limb or the unaffected portion of the injured one (loss of epicritic sensibility). Within this area of loss of sensibility to light touch, to which we gave the name of "intermediate zone," the patient is able to appreciate the sharpness of a pin prick and to differentiate temperatures below 18° C. from those above 45° C., naming them correctly.

Division of a peripheral nerve produces a well-defined loss of epicritic sensibility, a smaller loss of protopathic sensibility with, as a rule, ill-defined limits. In many cases there is no loss of deep sensibility.

Complete division of certain nerve branches produces no objective change in sensibility, these are the musculospiral below the point at which its external cutaneous branches are given off, the radial and certain cervical anterior primary divisions.

After Results.—The area of loss of protopathic sensibility may undergo some slight diminution, although no union with its own central end has taken place. This is due to reunion with the central nervous system throughout branches of cut nerves in the surrounding tissues, and not as was at one time suggested to the encroachment of nerve fibers from surrounding areas. After the freeing necessary before secondary suture, the loss of sensibility becomes as extensive as it was immediately after division.

The skin becomes thin, red, and shiny, and the fingers taper through wasting of their "pulp." These changes may, to a certain extent, be prevented by systematic massage. Ulcers may appear as the result of injury so slight that a neighboring sound part is not affected by the same trauma, but after complete division they do not arise spontaneously.

Nails become altered in texture and lose their gloss, but their rate of growth is not altered except as the result of interference with voluntary movement.

Motor Symptoms.—The detection of the paralysis may need careful examination, for the movements with which the contraction of the affected muscles is usually associated may be performed by other muscles. This has led to many cases of injury being overlooked and is not yet fully appreciated. Swan in 1834 recognized this and wrote "I . . . concluded that misconceptions have arisen from considering the general motion of the limb as evidence of the restoration of the nerve."

Létiévant laid great stress on this imitation by unaffected muscles of the movements usually performed by those paralyzed (*motilité supplée*).

This most often leads to difficulties in injuries of the median and ulnar nerves at the wrist; paralysis of the abductor and adductors of the thumb, of the opponens pollicis, and of the interossei may be easily overlooked.

Unless care be taken the affected muscles rapidly waste and contractions develop as the result of the overaction of opposing muscles.

The affected muscles cease to respond to stimulation with the interrupted current applied over their motor point in from four to seven days. By about the tenth day a change has occurred in the response given to stimulation with the constant current; the stimulus necessary to produce it is greater than on the sound side, the contraction so produced is sluggish and spreads in a wave-like manner from the spot stimulated. It is more easily produced when the anode is the testing electrode (polar reversal). These changes are spoken of as the "reaction of degeneration."

The length of time after separation from the central nervous system that the power of reacting to a constant current is retained varies within wide limits. It will depend upon the means taken to keep up their nutrition, and also perhaps to reunion with the central nervous system by means of "wound fibers." I have seen a patient in whom the muscles supplied by the musculospiral nerve reacted to the constant current twenty-three years after complete division.

Symptoms of Incomplete Division of Peripheral Nerves.—Under the term "incomplete division" those cases are included in which conduction is interrupted without the production of degeneration in the whole of the peripheral end of the nerve. It has been found, both clinically and experimentally, that a considerable portion of the trunk of a nerve may be divided without producing any change or one of a transient nature only. This is most important in connection with the operation of nerve anastomosis. I have on several occasions divided one-third of the internal popliteal nerve without producing any recognizable effect on motion or sensation.

But there are exceptions to this rule; it applies only to the trunk of a nerve well above the point at which branches are given off; if the incision cuts into the nerve just above the point of origin of a branch, the signs of complete division of that branch are produced. This is confirmed by the experiments of Bruandet and Humbert who found that the fibers in a peripheral nerve which go to make up any branch do not become grouped together until just before it leaves the parent trunk. In certain situations also—for example, in the anterior primary division of the fifth cervical nerve—the nerve fibers are arranged in a well-defined order, and incomplete division of this nerve may entail complete division of these motor fibers which supply the spinati and deltoid muscles. Again, in the trunk of the great sciatic nerve the external and internal popliteal nerves remain separate, hence incomplete division of the great sciatic may cause complete division of the external or internal popliteal nerve.

But in accidental wounds of nerves, in addition to the incomplete

anatomical division there is usually physiological division, the result of the transient compression of the intact nerve fibers by the cutting instrument or effused blood.

Absence of symptoms in many cases is due to the fact that more nerve fibers are present in the trunk of a nerve than are necessary to the supply of the part. When symptoms are present the recovery of function is due to restoration of conduction in the fibers which have suffered an incomplete physiological division. Those fibers which are separated from their nerve centres must of course degenerate and regenerate before they can again carry on their functions. It must, however, be remembered that the injury to the anatomically intact nerve fibers may be so great that complete physiological division is produced; this may also arise at a later period as the result of compression by fibrous tissue.

Sensory Symptoms.—In many cases there is no loss of sensibility that can be marked out by our present methods of investigation. Usually the patient is conscious of an area of skin altered in sensibility, and it is often possible to demonstrate this by the changed sensibility produced at its borders when a piece of cotton-wool or the point of a needle is dragged lightly across the skin from sound to affected portions (line of change). If the area of changed sensibility is well marked, response to the compass test will be defective.

In cases of greater severity the loss of sensibility to cotton-wool may be absolute, with borders as well defined as after complete division.

When the injury is more severe, impairment or loss of protopathic sensibility results and the sensory loss may resemble exactly that seen after complete division.

Motor Symptoms.—It was for long the recognized teaching that incomplete injuries of nerves affected the motor more than the sensory fibers. I showed in my Erasmus Wilson Lectures that this was not the case.

Paralysis of some or all of the muscles supplied by the injured nerve may result from incomplete division. It is only after sufficient time (eight to fourteen days) has elapsed, to allow of the development of electrical changes, that the diagnosis of incomplete division can be made apart from exploration.

In the least severe cases the muscles, though paralyzed, retain their irritability to the interrupted current. Usually the reactions that I consider typical of incomplete division are present. On about the tenth day after the injury the muscles do not respond to the interrupted current, but react in a characteristic manner when stimulated with the constant. The strength of current necessary to call forth the contraction is less than on the sound side; the contraction so produced is brisk as compared with that seen when the reaction of degeneration is present, and polar reversal is absent.

After Results.—Pain is a more frequent symptom than after complete division, and is often accompanied by tenderness of the skin (hyperalgesia), sometimes by glossy skin and changes in the growth of the nails.

These symptoms are irritative; they rarely arise immediately, a latent period of a few days to three weeks being present. The pain is most severe when there has been an incomplete anatomical division, and is most often seen after gunshot wounds.

Paget was the first to record a series of cases of this nature, but Weir Mitchell, Morehouse, and Keen gave the first complete description of this condition, named by them "*causalgia*," based upon their observations of gunshot wounds of nerves during the American Civil War. The following from Weir Mitchell's book describes the condition with accuracy: "The skin affected in these cases was deep red or mottled, or red and pale in patches. The subcuticular tissues were nearly all shrunk, and where the palm alone was attacked the part so diseased seemed to be a little depressed and firmer and less elastic than common. In the fingers there were often cracks in the altered skin, and the integuments presented the appearance of being tightly drawn over the subjacent tissues. The surface of all the affected parts was glossy and shining, as though it had been skilfully varnished. Nothing more curious than these red and shining tissues can be conceived of. In most of them the part was devoid of wrinkles and perfectly free from hair. Mr. Paget's comparison of chilblains is one we often used to describe these appearances, but in some instances we have been more strongly reminded of the characters of certain large, thin, and polished scars."

But, as seen in civil life, the condition is rarely so severe; it may result from penetrating wounds, primary injury in association with fractures, or a direct blow. After a latent period of a few days intense burning pain makes its appearance. The painful area is usually extremely tender and maps out the "full" distribution of the injured nerve.

Loss of sensibility may be present, varying according to the nerve injured and the degree of that injury; usually the loss is of epicritic sensibility only.

The skin sweats profusely and the affected area may often be marked out by beads of moisture. In some cases the subcutaneous tissue appears to be increased in size, and the nails may become more curved and grow faster than those of the unaffected hand. Blisters may make their appearance and break down to form ulcers, not only over the area of sensory loss, but often over the area in which there is hyperalgesia but no loss of sensibility.

In cases of incomplete division without irritation the changes in the skin are little marked unless the injury has resulted in trophic loss, when they may resemble those seen after complete division.

Method of Recovery.—After complete division of a nerve its whole peripheral end degenerates before regeneration can take place. There is no exception to this rule; all modern experimental and clinical investigation is opposed to the conception of "*primary union*." All the recorded "*instances*" of this can be explained as incorrect inferences drawn from the observed facts, as was first demonstrated by Létiévant, who explained that the so-called early recovery of sensibility and

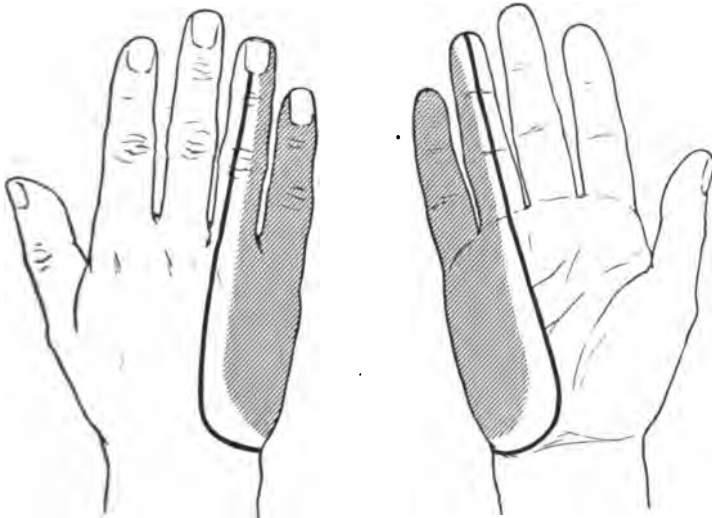
motion after suture was due to want of appreciation of the phenomena which follow division.

After complete division of a nerve followed by suture, an interval elapses before restoration of function commences. This varies with the age of the patient, the nerve injured, the method of healing of the wound, and the variety of suture, being more rapid in youth and after primary suture, and retarded by suppuration.

Sensory Recovery.—Sensory recovery may be divided into three stages:

1. Restoration of protopathic sensibility.
2. Restoration of sensibility to light touch and minor degrees of temperature.
3. Restoration of the power of localization.

FIG. 2



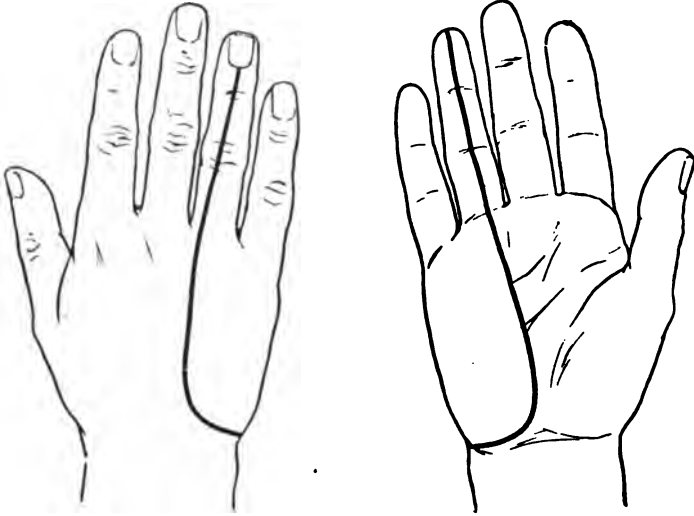
Loss of sensibility after complete division of ulnar nerve.

In from six to sixteen weeks the first stage commences. The area of protopathic loss diminishes in extent and this form of recovery is complete in from four to twelve months after suture. This is the first stage of recovery (see Figs. 2 and 3). During this stage blisters may appear spontaneously over the analgesic area, but on its completion all ulcers heal and no further blisters make their appearance.

During the whole of the first stage the area of loss of sensibility to light touch is unchanged. Gradually it diminishes in extent from its proximal border until, at a time varying from about twelve months to eighteen months, the whole of the affected portion is sensitive to light touch and the intermediate degrees of temperature. This concludes the second stage (Fig. 4).

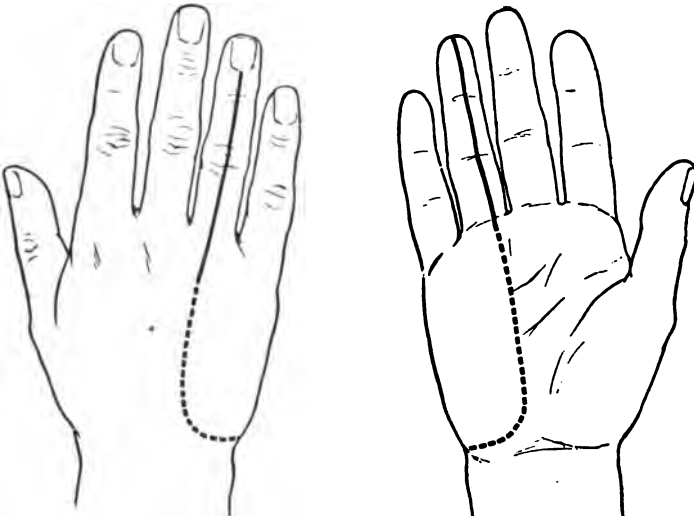
The interval between the end of the first and the beginning of the second stage varies with the variety of suture and the distance from

FIG. 3



End of first stage of recovery.

FIG. 4



Commencement of second stage of recovery. The dotted line marks the area regaining sensibility to cotton-wool.

the periphery of the point of section. After division of the median or ulnar nerves at the wrist, followed by primary suture, an interval of

more than about six weeks is unusual unless suppuration has occurred. In favorable cases in which the correct after-treatment has been faithfully carried out it may be inappreciable. If suppuration occur, or secondary suture has been performed, the commencement of the second stage is much delayed and recovery may be permanently arrested.

It has long been the teaching that the distance from the periphery at which a nerve is divided affects the time necessary for recovery. Taking recovery as a whole, this is in accordance with my experience, but it does not apply to the commencement of the first stage of sensory recovery. The distance from the periphery at which a nerve is divided does not affect the interval between suture and the commencement of the first stage, but it markedly prolongs the interval between the end of the first and the beginning of the second stage, and final recovery is much delayed.

After restoration of sensibility to light touch and the minor degrees of temperature the sensory condition of the part is by no means normal. If a pin be dragged across the skin from normal to affected parts, sensation is found to change as soon as the old boundary for the loss of light touch is reached. While this area of changed sensibility is present the discrimination of two points (the compass test) is always defective. Improvement in the power of accurate localization constitutes the third stage of recovery. No sensory recovery should be recorded as perfect unless the appreciation of the compass test is as good as on the sound side. Until this has taken place the part is useless for delicate work.

It has been suggested that this third stage of recovery represents the time during which reëducation of the centre is taking place. After division and suture it can rarely happen that the nerve fibers in the central end reëstablish connection with those fibers and end organs with which they were originally in continuity. Osborne and Kilvington, as the result of experimental work on axon bifurcation, have shown that regenerating motor fibers can divide into at least two separate and distinct axons.

Motor Recovery.—At a time varying with the distance of the injury from the periphery and the age of the patient the muscles regain their voluntary power. This return is usually preceded by a change in the electrical reactions of the affected muscles; they become identical with those I have described as typical of incomplete division. Head and the writer found that irritability to the interrupted current is usually present on the same date as the first return of voluntary power is noticed.

Controversy has raged over the question of the method of regeneration after section of a nerve. Of recent writers on the subject, Mott, Halliburton and Edmunds, Langley and Anderson, Ramón y Cajal, Marinesco, and Howell and Huber, have supported the theory that regeneration takes place by the downgrowth of axis-cylinders from the central end into the nerve sheaths of the peripheral end.

Ballance and Purves Stewart, Kennedy, Bethe have been prominent upholders of the theory that the new axis-cylinders are developed

in situ as the result of the action of the neurilemma cells and become secondarily connected with the central nervous system.

All recent writers are agreed that with or without union with the central system, proliferation of the neurilemma cells leads to the formation of a strand of spindle-shaped fibers called "embryonic nerve fibers," "band fibers," "neuroplastic fibers." Certain observers, among whom are Ballance, Purves Stewart, Bethe, and Langley and Anderson, have found medullated axis-cylinders developed in peripheral ends entirely separated from their own central ends. The former observers consider this as evidence of autogenetic regeneration, but Langley and Anderson showed conclusively that it was due to union with the central nervous system through nerves in the tissues around. All the new medullated fibers degenerated when the nerves which ran to the tissues near the cut end were divided close to the spinal cord. Harrison's experimental embryological work shows that the developing nerve fiber is of central origin.

It seems certain that no regeneration takes place in the peripheral end of a divided nerve without union with the central nervous system.

After incomplete division of a mixed nerve the loss of sensation and motion may at first resemble that which follows complete division, but the method of recovery is entirely different.

After complete division of a nerve and suture, sensibility to prick is restored before the commencement of recovery of sensibility to light touch. Complete sensory recovery often occupies several years.

But after incomplete division sensibility to light touch and to prick are restored together (see Figs. 5 and 6), and, unless nerve fibers have been anatomically divided in considerable number, the power of appreciating two points (the compass test) is soon regained. This is an extremely important point, for upon the recovery of this power of localization depends the utility of the part for fine work. It is important to recognize that in injuries of nerves without interruption of their anatomical continuity the power of localization returns quickly—unless the injury has been sufficient to cause complete division, in this case the usual three stages are present, but the time of the third stage is much shortened.

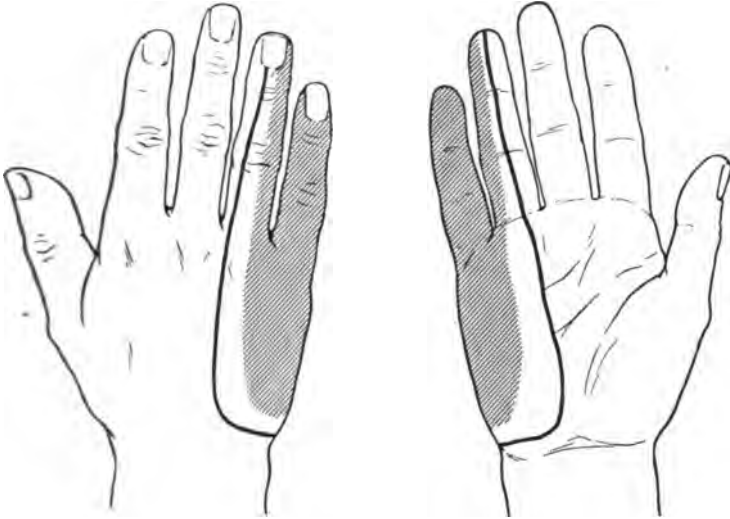
Knowledge of this method of sensory recovery, first described by Head and the writer, is a valuable addition to our powers of diagnosis. If both forms of sensibility are recovering together, it is certain that the injury has not been severe enough to produce complete interruption of conduction in the injured nerve, with degeneration of the whole peripheral end.

Motor recovery after incomplete division follows the same march as after complete; the muscles nearest the seat of the injury first regain voluntary power and excitability to the interrupted current.

In the cases in which the reactions typical of incomplete division are present, voluntary power usually returns before the reestablishment of excitability to the interrupted current.

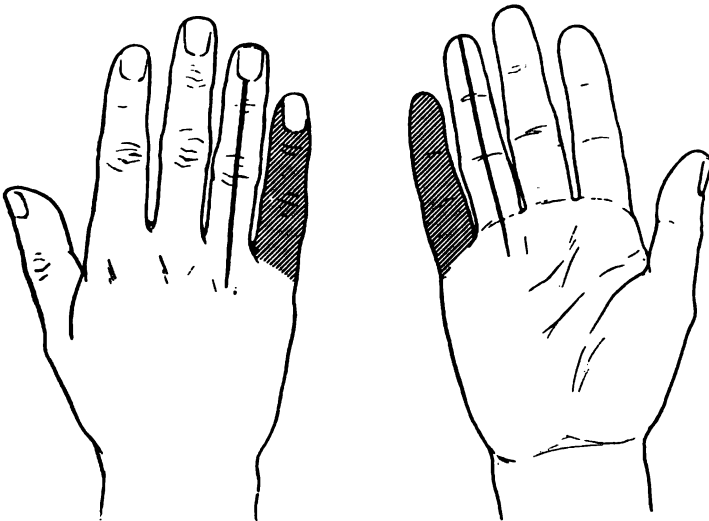
Sensory recovery usually begins in about three weeks and is complete in about six months. Motor recovery in from a few days to ten weeks.

FIG. 5



Loss of sensibility after complete division of ulnar nerve.

FIG. 6



Showing method of recovery after incomplete division.

These times of motor and sensory recovery are approximate only, and vary with the severity of the injury and its distance from the

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periphery. When epicritic sensibility alone is lost recovery is much more rapid than when both forms of sensibility are affected. When the injury affects the brachial plexus, considerably longer time is necessary for the commencement and progress of recovery.

To sum up, after incomplete division of a mixed nerve, both forms of sensibility (epicritic and protopathic), if lost, return at the same time, commencing at a date which varies with the distance of the injury from the periphery from about three weeks at the wrist to six months in the plexus, and also with the degree of the injury. Complete recovery, as a rule, rapidly ensues. Muscular recovery commences at a time which varies in the same way. In cases in which the muscles, though paralyzed, retain their irritability to the interrupted current, recovery commences in three or four weeks, sometimes earlier, and soon becomes perfect. This degree of injury is seen most often as the result of compression of the musculospiral nerve, producing sleep, anesthetic or crutch paralysis. If the reactions typical of incomplete division are present a much longer time is necessary.

After neurolysis, or when the nerve has been relieved from any form of pressure, recovery follows exactly the same lines.

Method of Examination.—It is necessary to follow some definite plan or important points are omitted. Diagnosis consists in the discovery of the nerve or nerves injured, the position of the injury and its nature, whether complete or incomplete.

The history should first be taken. Important points to be elicited are: the date of the accident and its exact nature; what symptoms pointing to a nerve injury first attracted the attention of the patient, and the time after the accident they were noticed.

In old cases inquiry should be made for increase or diminution in the extent or degree of the sensory or motor symptoms. If pain has been present at any time, questions must be put to ascertain the date of its onset, its exact distribution and character, if it varies in severity from time to time, if it has spread to areas other than that first affected, or the patient is aware of anything that increases it or can obtain relief from it in any way. Some idea must be formed of its severity, whether keeping the patient awake at night, or affecting his mental condition or general health. If the nerve was injured in a wound, how long the wound took to heal, and the nature of its treatment.

It is useful to conduct the routine examination under the following three headings: (1) General inspection of the part injured; (2) examination of sensation; (3) examination of muscles.

1. **General Inspection of the Part Injured.**—(a) Position of injured part or limb. (b) Wounds, scars, etc. (c) Condition of skin: changes in color, desquamation, blisters, ulcers, alterations in temperature. (d) Condition of nails and hair.

2. **Examination of Sensation.**—(a) For tenderness. (b) For loss of epicritic, protopathic, and deep sensibility.

3. **Muscular Examination.**—(a) Wasting, general and localized, contractures. (b) Paralysis. (c) Electrical changes.

1. (a) The position taken up by the injured limb or part may point to the nerve or nerves involved; the drop-wrist of musculospiral injury, the drop foot of injury to the external popliteal, the ulnar hand and the true "claw hand" of injury to the median and ulnar, inner cord of the plexus or first dorsal root are examples.

(b) The nature and anatomical position of wounds and scars must be noted. If a scar, whether showing signs of primary union or of healing by granulations, whether free of the deep tissues, or, if adherent, to what structures? Palpation in the neighborhood may reveal the presence of bulbous enlargements, or of tenderness accompanied by pain referred to the affected limb.

(c) The condition of the skin should always be investigated; the desquamating skin seen a few days after the injury, the dry, bluish-pink, atrophic skin of a later period or the true "glossy" skin, often covered with beads of perspiration, will all aid in diagnosis. When blisters are present, their exact situation and relation to the area of protopathic or of epicritic loss. A note should be made as to the mode of onset of the blisters or ulcers, if they originated in response to injury or appeared spontaneously. The latter may point to commencing recovery.

(d) The nails should be examined for changes in color or gloss, for brittleness, growth of epithelium under the free edge, curvature, ridging, etc.

2. In testing sensation the patient should be comfortably seated with the affected part resting easily, so that no restraint is imposed or muscular effort necessary to maintain its position. The eyes of the patient should be closed, and he should be told to speak whenever he feels anything, whether a prick, a touch, or any other sensation or change in sensation. The usual method of testing, touching, or pricking the patient and saying "do you feel this, etc.?" is more time-consuming and quite untrustworthy.

(a) If pain is complained of or the condition of the part is suggestive, the examination should be first conducted to find out if tenderness of the skin is present and its exact extent marked out. This can be done by dragging the point of a pin lightly across the limb from the sound to the affected side, the patient being told to speak as soon as the stimulus becomes painful.

(b) The condition of epicritic sensibility should be first investigated.

For routine clinical work the testing of light touch is usually sufficient; but temperature tests and the compass test should be employed in any difficult or doubtful case. Light touch is tested by means of cotton-wool rolled up to form a pledget, or a soft camel's-hair brush stroked gently over the affected part. This test must be applied carefully. If used roughly, or applied at right angles to the surface, deep touch may be evoked; even when lightly employed over desquamating areas this stimulus may be appreciated by means of deep sensibility.

Errors may also arise over hair-clad parts in which there is retention or return of sensibility to prick. The sensation produced by cotton-

wool in these cases is different from that given by stimulation of the hairs on a normal part of the limb with cotton-wool; it possesses the radiating, tingling character associated with protopathic sensibility. On shaving a part in this condition it becomes entirely insensitive to cotton-wool. This should be done in all cases of doubt and the temperature and compass tests applied.

Glass test-tubes containing ice and water at 50° C. are used for investigating the extreme degrees of temperature. For minor degrees similar tubes containing water at about 24° and 38° C.; these temperatures should be readily discriminated by the patient as cool and warm over the corresponding sound part. So many difficulties surround the testing of the minor degrees of temperature that too much reliance should not be placed on failure to discriminate.

In applying the compass test the blunt points of a pair of compasses are separated from one another for a measured distance. The skin of the affected portion of the limb is touched, and the patient is asked to say after each stimulation whether he has been touched by one or two points. When they are separated for less than a certain distance, varying with the part of the body under examination, the points no longer appear as two on the normal skin. Head and the writer found that two points could be accurately recognized over any part of the normal palm when separated for 1 cm. and applied transversely. In carrying out the test the method introduced by us is useful. The patient is touched ten times with one point, ten times with two, each being applied at random. The results are recorded graphically in the following manner: Every time the patient's answer is correct a stroke is made, above a horizontal line if he was touched with one point, below if he was touched with two. An incorrect answer is recorded by a cross. Thus, if he answers one when touched with two points a cross is placed below the line. A preceding stimulus frequently has an effect upon those which follow it, and to register the order in which the stimuli have been applied is therefore an additional aid to the interpretation of the records. Thus, if the testing began with four double touches correctly answered four strokes would be made below the line. At the point above the line directly over the last of these would begin the record of the subsequent single stimuli; in this way the results of all further stimuli are recorded until the number is complete.

Perfect appreciation of the compass points at a distance of 2 cm. would be represented thus:

2 cm. $\frac{1}{2}$ $\frac{\text{III}}{\text{III}}$ $\frac{\text{II}}{\text{III}}$ $\frac{\text{IIII}}{\text{III}}$

If, however, the patient is unable to differentiate the two points at this distance, answering one to every stimulation, the record would stand:

2 cm. $\frac{1}{2}$ $\frac{\text{III}}{\text{XX}}$ $\frac{\text{II}}{\text{XXX}}$ $\frac{\text{III}}{\text{X}}$ $\frac{\text{IIII}}{\text{XXXX}}$

Such a formula would show that when 2 cm. apart the sensation produced by two points is well below the threshold at which discrimination becomes possible. Less complete failure would be represented by some such formula as:

$$2 \text{ cm. } \frac{1}{2} \frac{\text{I I X X}}{\text{X I}} \frac{\text{I X}}{\text{I X}} \frac{\text{I X X I}}{\text{I I X X X X}}$$

where 50 per cent. of the answers are wrong with one point, 60 per cent. with two points. A curious phenomenon is the tendency to appreciate one point as two as soon as the limits of accurate discrimination are passed.

Diagnosis.—In injuries of the cervical and sacral regions of the spinal cord and in hysteria following injury, difficulty may be experienced.

Spinal Cord.—In affections of the spinal cord there is not only interference with the conduction of impulses, there is also the local effect of destruction or interference with the function of the segment affected and the fibers entering it at this level.

Motion.—A lower segment lesion produces a flaccid paralysis accompanied by changes in the electrical excitability of the affected muscles, and if complete, the reaction of degeneration. It should be remembered that motor fibers may be affected at their anterior horn cells as the result of injury or anterior poliomyelitis, in the anterior root or peripheral nerve. In all these situations motion may be affected without change in sensibility.

The grouping of the affected muscles may at once denote the peripheral or central position of the lesion. For example, the deformity produced by an injury of the ulnar nerve is different from the true claw hand produced by a lesion of the first dorsal root or segment; paralysis of the extensors of the fingers and thumb and the ulnar extensor of the wrist, while the supinator longus and radial extensors of the wrist remain unaffected, at once denotes the root or central position of the lesion.

The Erb-Duchenne or the peroneal group of muscles may suffer as the result of interference with their supply in the anterior horn root or peripheral nerve. In the former case (Erb-Duchenne) the diagnosis may rest entirely upon the history, for section of the anterior primary division of the fifth cervical nerve, the nerve supplying this group, does not produce any sensory loss. In the latter case, if the lesion is in the root or anterior horn cells, the tibialis anticus often escapes and there is no loss of sensibility—an impossibility if the motor affection were due to injury of the external popliteal nerve which supplies these muscles.

It is impossible to diagnosticate by symptoms alone between a lesion of the anterior horn cells and of an anterior root. The necessity for such a diagnosis fortunately does not often arise, and when it does the history of the case usually makes it clear.

Paralysis, the result of an upper segment lesion, is easily distin-

guished by the electrical reactions of the affected muscles remaining unchanged and, in most cases, by the spasticity present.

Sensation.—It was shown by Head, Rivers, and the author that the afferent impulses are grouped in an entirely different manner when the spinal cord is reached. The tracts in the spinal cord are devoted to the conduction of impulses concerned with pain, heat, cold, and touch; it is no longer a question of epicritic, protopathic, and deep sensibility.

This subject has been fully worked out by Head and Thompson, and the following is drawn from their article on the subject.

Pain.—After division of a peripheral nerve or posterior root, those parts only become insensitive to the pain of deep pressure which are at the same time totally insensitive to the tactile element of the stimulus. Unless all deep sensibility be abolished pain will be caused by excessive pressure. But if the lesion lies within the spinal cord sensibility to pain is abolished as a whole whatever the form of stimulation.

Heat and Cold.—When the lesion is within the spinal cord sensibility to heat may be abolished without sensibility to cold. When sensibility to heat is abolished in consequence of an intramedullary lesion, the patient no longer appreciates any warm or hot stimulus; in the same way, when sensibility to cold is abolished the patient no longer appreciates any cold or cool stimulus. All distinction between the minor and the extreme degrees of temperature is lost—the appreciation of heat or of cold is lost as a whole. The patient may be insensitive to all degrees of temperature and yet be able to appreciate the lightest touch and discriminate the points of a pair of compasses—conditions which can never occur from a lesion of a peripheral nerve only.

Touch, Superficial and Deep.—After division of a nerve or posterior root, light touches with cotton-wool are usually not appreciated, though deep touch (pressure) evokes a response. But when the lesion lies within the spinal cord both forms of touch are affected together.

Passive Movement and Position.—After division of peripheral nerves the recognition of passive movement and of the position into which any part of the limb has been placed (passive position) is associated with the integrity of deep sensibility. But with an intramedullary lesion it is entirely dissociated. The patient may be able to appreciate passive position and movement although totally insensitive to every other sensory stimulus, or *vice versa*. In a similar way, a patient may be able to appreciate all varieties of touch perfectly, and yet be unable to discriminate two points (compass test). In lesions of peripheral nerves the compass test is always affected with light touch.

A rearrangement of impulses takes place within the spinal cord and their interruption causes loss of sensibility to pain, heat, cold, or tactile sensibility as a whole instead of to epicritic, protopathic, and, in some cases, deep sensibility as occurs when the continuity of a peripheral nerve is interrupted.

Put briefly the important points are as follows: After division of a peripheral nerve or of posterior roots there may be loss of epicritic,

protopathic, and deep sensibility. After division of a peripheral nerve the loss of epicritic sensibility is greater than the loss of protopathic; after division of posterior roots, the loss of protopathic sensibility exceeds in extent the loss of epicritic. But when the injury affects the spinal cord, pain, temperature appreciation, touch, may be affected separately. Usually light and deep touch are well recognized although sensibility to pain and to temperature is absent.

In unilateral lesions of the spinal cord the appreciation of pain, heat, and cold is affected on the side opposed to the lesion, passive movement and position on the side of the lesion and the motor affection.

Hysterical Affections.—This type of functional nervous disorder may follow any form of injury; thus I have seen anesthesia and paralysis of the whole hand follow a burn of the thenar eminence in a woman, and a fracture of the radius in a boy of twelve. It may complicate recovery from operation and gives rise to much difficulty when complicating a nerve injury.

While it may follow an injury to either sex it is more often seen in the male. So far as I have been able to ascertain they are usually healthy individuals and may show no other hysterical manifestation.

As a rule loss of sensibility and paralysis are both present, but either may be found alone, the former more often than the latter. The loss of sensibility is to all forms equally (including deep touch) a variety of loss that does not occur after any peripheral nerve, posterior root, or spinal-cord injury; its upper limit usually surrounds the limb, often at the level of a joint, and all forms of sensibility are lost up to the same level. In the upper limb the loss of sensibility may cover the pectoralis major muscle in front and the scapula behind (fore-quarter type).

The paralysis may persist unchanged for years and marked muscular wasting will then occur. It is, as a rule, flaccid, and no attempts are made to throw the affected muscles into action, but occasionally a patient is met with in whom attempts to perform a movement—for example, flexion of the elbow—cause an equal and simultaneous contraction of both flexors and extensors, rendering the diagnosis easy. Contractures may be present, differing from those seen as the result of injuries to nerves, in that all the muscles are affected, not only those on the same side of the limb as the contracture. For example, in a contracture at the elbow of a hysterical limb, not only are the flexor muscles rigid, but any attempt to further flex the forearm is met by contraction of the triceps.

No difficulty should arise in the recognition of most examples of this condition; the loss of sensibility is diagnostic and the flaccid paralysis with retention of electrical reactions typical. But when complicating a nerve injury it gives rise to difficulty. It explains many of the recorded cases of nerve "concussion" in which paralysis of the whole of a limb results from a gunshot injury which may or may not have injured one nerve. In the latter case the widespread symptoms rapidly clear, leaving signs of involvement of one definite nerve.

In civil practice I have seen several examples of this condition some

time after the original accident, but have not yet observed it at the time of infliction of the nerve injury.

Careful attention to symptoms will enable the diagnosis to be made. The paralysis may be widespread and affect muscles central to the site of the nerve injury, but occasionally—for example, after division of the median or ulnar nerves at the wrist—all the intrinsic muscles of the hand are found to be paralyzed, and only the electrical examination reveals the functional nature of the paralysis of one group of these. In testing sensibility the remarkable correspondence of the upper limit to all forms of sensibility and the affection of deep sensibility should make the diagnosis, even in these cases, easy.

TREATMENT

The importance of early diagnosis and correct treatment of injury to one of the nerve trunks of a limb is impossible to overestimate. Injury of the median, ulnar, or musculospiral nerves, unless completely recovered from, leaves the patient permanently unfitted to follow any employment involving the full skilled use of that limb. Prognosis depends upon prompt treatment at the time of the accident and efficient supervision for many months after.

The treatment of injury to a nerve in its continuity consists in keeping up the nutrition of the parts supplied by it and preventing the occurrence of contractures until conduction is restored by natural means alone or aided by operation. It must be remembered that in comparatively few cases is operation necessary, it should always be avoided if possible and sufficient time given to be certain that recovery will not ensue unaided. Even when operation is undertaken every care should be used in cases of physiological division to avoid nerve section, this must only be done if it is inevitable.

It is essential to maintain relaxation of the paralyzed muscles until voluntary power is restored; unless this is done recovery may be indefinitely delayed. I showed a striking example of this: a male, aged twenty-six years, injured the musculospiral nerve two and a half years before he came under my care. When I saw him in March, 1909, all the muscles supplied by the musculospiral nerve in the forearm were paralyzed and gave the "reactions of incomplete division." He had been going about with his hand hanging down. I had the limb placed on a splint with the fingers extended and the wrist hyperextended; the condition began to improve at once. Within a week the muscles responded to stimulation with the interrupted current. Within three weeks the extensors of the wrist and fingers acted voluntarily.

Unless guarded against, contractures will occur in the opponent muscles and may effectually prevent complete recovery. For example, if "claw hand" is allowed to develop after an injury to the ulnar nerve it is extremely unlikely that perfect recovery will ever ensue.

This muscular relaxation is the most important point in treatment,

but it is the one most often neglected. It is quite useless to undertake the treatment of a case of nerve injury unless this can be carried out.

The splint or retention apparatus should be removed daily if possible, and massage should be regularly employed with electrical stimulation of the affected muscles with whichever current they will respond to, this latter is not of such great importance as the systematic massage. As soon as voluntary power is restored to any muscle it should be systematically exercised every day.

In cases needing operation it is too often assumed that when once the ends of the nerve have been united the surgeon's work has ceased; it has only just begun. The careful supervision of the patient and the direction of the treatment, it may be for two or three years, is most important.

No work should be attempted with the damaged limb. In complete injuries the part must be protected against cold and the patient warned that this, and injuries so slight that they have no effect upon sound portions of the limb, may cause blisters upon the affected side; if these become infected serious consequences may result.

Operation upon Nerves.—There are a few general points that should be first mentioned. Incisions must be of sufficient length, any attempt to work through small incisions will lead to more damage being done to the nerve. A tourniquet should not be used; apart from the danger of producing a fresh nerve injury it is well to have the wound as dry as possible; considerable oozing is inevitable after the removal of a tourniquet.

The nerve should always be exposed above and below the seat of the injury; attempts at finding the nerve at the actual seat often lead to considerable damage being inflicted.

Nerves must be handled with the utmost gentleness and not lifted on hooks or forcibly pulled on one side. The whole nerve should never be grasped in the forceps, it should be steadied by picking up its sheath with fine-toothed forceps.

Incisions into nerves or freshening their ends must be done with a sharp scalpel: scissors should not be used for the purpose, as they crush the nerve and render regeneration less likely.

Absorbable material should always be used for sutures. I prefer No. 0 Van Horn catgut; if there is any tension it should be twenty-day chromicized. Silk or thread must never be used; not only may it prevent complete regeneration by interposing a foreign body in the substance of the nerve, but it occasionally leads to neuritis and the formation of an intraneural abscess months or years later. I have recorded several such cases.

Whenever a nerve has been freed it should be protected by wrapping with cargile membrane. If this is not at hand it can be surrounded by an excised portion of one of the superficial veins of the patient.

Wounds, especially those in the region of the wrist, should be closed in layers, and where, as in this position, the limb has a fascial covering this should be carefully closed or hernia of the tendons may result.

Nerve Injuries in Wounds.—In cases of accidental wounds the possibility of nerve injury must be borne in mind, and a careful examination conducted before any attempt is made at treatment. This rule is not infrequently neglected, especially in the common glass cut wounds in the region of the wrist, with the result that nerves are left ununited or sutured to tendons. Nerve injuries are overlooked not from a want of knowledge, but as a result of imperfect examination. It is not necessary, in fact, it is usually impossible, in these cases to discover whether the nerve injury is complete or incomplete, all that is essential is to know that a nerve has been injured and then at exploration to examine that nerve. If this rule were remembered secondary suture would become what it should be—an almost unknown operation.

When the fact of nerve injury in an accidental wound has been established, the incision necessary for its exploration should be so planned that it can be easily examined. If it be found incompletely divided the gap should be brought together with one catgut stitch, and then surrounded with cargile membrane to prevent ingrowth of fibrous tissue and adhesion to surrounding structures. If the nerve is completely divided the ends, if lacerated, should be trimmed with a sharp scalpel. If the nerve is divided at two or more levels a portion being loose, this should be sutured in. If so much is destroyed that the ends cannot be brought into apposition, one of the methods described later must be adopted.

The catgut used should be as fine as possible and passed with a small round needle through the whole thickness of the nerve and tied with just sufficient force to bring the ends into apposition. If the nerve is a large one, another suture may be passed at right angles to the first. The greatest care must be taken to avoid longitudinal rotation of either end of the nerve, we should endeavor to bring the axis-cylinders in the central end of the nerve opposite their own severed ends in the peripheral portion.

It is wise in most cases to insert a drain for twenty-four hours.

After suture of the skin wound the limb should be placed on a splint arranged to relax the paralyzed muscles and prevent tension on the nerve junction.

Recovery after Primary Suture.—Perfect recovery is possible after primary suture; perfect function may be restored to the affected muscles and no difference noticed by the patient in the sensation of the part as compared with the corresponding sound one. Although possible, and I have seen it follow primary suture even of the ulnar nerve, it is unusual. Unless the wound healed by first intention, and the after-treatment has been efficient, it cannot be expected in a mixed nerve.

The prognosis will depend to a certain extent upon the nerve injured; for example, the musculospiral nerve in the lower third of the arm carries no exclusive supply to any portion of skin, and the muscles it innervates are not so intimately associated with delicate movements of the fingers as are those supplied by the ulnar. Complete recovery is reached more quickly than in other nerves.

The further the seat of injury from the periphery, the longer the time necessary to full recovery and the less likely is it to occur.

I have personally observed over 60 cases of primary suture. In all motor power was regained and the second stage of sensory recovery completed.

After primary suture of one of the nerves of the forearm, uncomplicated by suppuration, recovery will ensue and ultimately become perfect if appropriate after-treatment is adopted. Muscular power will be restored in about nine months according to the lesion, but perfect sensory recovery must not be expected under three years.

Treatment of Subcutaneous Injuries.—As the result of subcutaneous injury (by pressure or traction) all the immediate symptoms of complete division may develop. There is no sign that can tell us in these cases if the nerve is ruptured (anatomical division) or is in naked-eye continuity (physiological division). It may be impossible to make the diagnosis until ten or fourteen days after the injury, a time sufficient to allow for the development of the reaction of degeneration.

If seen at once the limb should be put at rest with the paralyzed muscles relaxed. Immediate operation should only be carried out when in a nerve injury complicating a fracture the signs point to complete interruption of conduction, and in injuries of the brachial plexus in adults.

Massage should be regularly employed, and the muscles electrically stimulated. At the end of a fortnight the electrical reactions should be taken. If at this date the muscles react to the interrupted current, or give the reactions of incomplete division, this treatment should be continued. Recovery can be confidently expected in the former case in a few weeks, in the latter in a few months. If improvement does not take place or the nerve has become secondarily involved, operation should be undertaken. It will usually be found that the nerve is compressed. It should be freed (neurolysis) and wrapped in cargile membrane; this latter is essential, many cases of relapse have been recorded when this has been omitted.

After neurolysis has been carried out recovery is usually rapid. In a case recently under my care in which the median nerve was compressed in the scar tissue resulting from a wound of the wrist three and a half months previously, the only sign before operation indicating incomplete division was the electrical reactions of the paralyzed muscles. When the stitches were removed eight days after neurolysis, both forms of sensibility had commenced to recover together, and within three weeks voluntary power had begun to return.

If the true reaction of degeneration has developed, the condition of the nerve should be investigated by exploration. If the nerve be found ruptured the ends should be freshened, suture performed, and the usual after-treatment adopted. It is in the cases in which the division is physiological that difficulties arise; the rules are difficult to formulate. It is a matter of personal experience to decide at operation between the cases that may be safely left and those in which resection of the

damaged portion is necessary. If on exploration the nerve appears little altered it should be wrapped in membrane and left. If in any doubt it is wiser not to resect.

Nerve Injuries Complicating Fractures and Dislocations.—The nerves most often injured as the result of fractures are the musculospiral in association with fractures of the middle and lower thirds of the humerus, the external popliteal in fractures of the neck of the fibula, the ulnar in fractures of the lower end of the humerus, and in rare instances the median in Colles' fracture.

The injury may be primary, the nerve being ruptured; lacerated, pressed upon by the fractured ends, or nipped between them. In secondary injury the nerve may become involved in callus or fibrous tissue, or as occurs especially in the case of the ulnar, suffer from the effect of the long-continued pressure of displaced bone.

Primary injury is more common than secondary, but unless definitely looked for at the time of the accident, symptoms are first discovered on removal of the splints.

If nerve injury is found when the recent fracture is examined, and this in the great number of cases will be the humerus with involvement of the musculospiral, operation should be undertaken without delay. At the same time it will usually be wise to treat the fracture operatively.

When the injury, as is so often the case, is discovered when splints are removed operation should be undertaken if the symptoms are those of complete division. The surgeon must be prepared to undertake nerve bridging, especially in old cases, and have fully decided what method to use. If, however, they are those of incomplete section, operation must not be considered, unless treatment for several weeks shows that recovery is not ensuing. If signs of nerve involvement develop with the use of the limb, operation should be undertaken at once. This consists in freeing the nerve from the pressure of callus or fibrous tissue and wrapping it in cargile membrane to prevent further implication. Unless this latter is carried out the nerve may again become involved.

Operation is rarely necessary in nerve injury associated with dislocations. The injury is seldom secondary and may be due to the injury producing the dislocation, or to the pressure of the head of the bone or faulty efforts at reduction. In unreduced dislocations of the head of the humerus the brachial plexus may suffer compression.

It is in recent dislocations of the humerus that injury most often occurs. The whole plexus may suffer, but it is most often the inner cord; in subglenoid dislocations it is the posterior cord or circumflex nerve.

In the reduction of dislocations of the hip, congenital or acquired, the great sciatic (usually its external popliteal division) and the anterior crural are those damaged. In forward dislocations of the head of the radius the posterior interosseous nerve.

Treatment proceeds on the usual lines. In injury of the posterior interosseous nerve from dislocation of the head of the radius, operation is indicated. When pain results from the pressure of the dislocated

head of the humerus it is usually necessary to free and wrap the nerves and remove the compressing head.

Secondary Suture.—By this I understand union of the ends of a divided nerve after degeneration has taken place in its peripheral end. It is a procedure that should become one of the rarest operations in surgery. It may be unavoidable in a few cases of subcutaneous injury, but it argues a serious want of care if medical treatment was sought at the time the nerve was divided in an open wound.

It is first necessary to consider how long after injury operation is likely to be followed by success. This is always an extremely difficult question to decide and depends upon many factors. If the nerve was divided in an open wound, the method of healing of that wound; nothing hinders recovery to so great an extent as suppuration in the original wound. The condition of the muscles as regards wasting and the retention of irritability to the constant current, the presence of contractures in opposing muscles and the condition of ligamentous structure surrounding joints. The condition of the hand of a patient, for example, who has suffered division of the ulnar nerve below its dorsal branch in whom a marked claw hand has developed is little likely to be improved by suture, although the muscles may regain their irritability to the interrupted current.

I believe that the time after the accident does not affect it directly. The divided peripheral end enters into the resting condition and any damage that is to occur in the spinal cord occurred early. Bowlby is also of this opinion.

It is extremely important to attempt to estimate the probable extent of recovery; from the motor standpoint if great wasting with contracture has occurred, as is so common after ulnar injuries, operation is not worth undertaking; but if trophic ulceration is present, operation will prevent this, for protopathic recovery is almost certain and ulceration, Head and the writer have shown, ceases on the restoration of protopathic sensibility.

Again, it is useless carrying out the operation unless the patient will give up sufficient time to treatment. For example, if the musculo-spiral is the nerve injured, the patient must be told that the part must be kept in a splint for six to nine months after operation. I never undertake operation unless I am assured that this can be carried out.

The steps of the operation are three:

1. Identification of the ends of the nerve.
2. Freeing and freshening the ends of the nerve.
3. Reestablishment of anatomical continuity.

1. The incision must be of sufficient length to expose the nerve well above and below the seat of the injury. Any attempt to find it directly at the seat of the injury will only lead, in most cases, to unnecessary damage being inflicted on the nerve.

2. The bulb with the fibrous tissue which is usually found surrounding and uniting the two ends should be well freed and the nerve stretched. After this has been done the bulb on the central end is

removed with a sharp scalpel. From the lower end only the fibrous upper extremity need be removed; the whole of the lower end of the nerve is in the same condition.

3. It often happens that the ends do not come readily into apposition; it was for this reason that the preliminary stretching was recommended. This will give fully an inch in the upper limb and, combined with relaxation of all the joints over which the nerve passes, will rarely fail to enable them to be brought into contact. If a gap is still left one of the methods described later should be adopted.

After closure of the wound the limb should be put up so that there is no tension on the nerve and the paralyzed muscles are relaxed. The position necessary to prevent tension on the junction must be maintained until the wound is soundly healed and then very gradually corrected.

Recovery and Prognosis.—Recovery is much slower and prognosis worse than after primary suture, but follows the same stages.

Considerable interest attaches to the commencement of the first stage of restoration of sensibility. From time to time instances of "rapid" return of sensibility after secondary suture have been recorded by many observers, among the more recent being Ballance and Kennedy. I have carefully examined for it after operation in 37 personal cases but have not yet observed it. My attention has more than once been drawn by hospital residents to the "rapid return of sensibility to prick," after secondary suture, which, on careful testing in the usual manner, proved to be deep sensibility. In one patient upon whom I had performed secondary suture of the median nerve it was said that sensibility to prick had returned on the day following operation. On testing, I found that he complained of pain on pressure, but could not distinguish the sharpness of the point of a pin; it was equally painful and produced the same sensation as pressure with the blunt end of a pencil; moreover, he was entirely insensitive to the painful interrupted current, and all temperature appreciation was absent. There was no doubt that the pain was that caused by deep pressure, which could be readily evoked before operation.

Before concluding that a rapid return of sensibility has taken place in any patient, sensibility must be carefully tested in all forms and mapped out on charts before and after suture, at first day by day, later week by week, up to recovery. It is evident, however, that a rapid return of sensibility is not to be expected after secondary suture, and that its "occurrence" must be regarded as unusual.

The time of commencement of the first stage of sensory recovery may be shorter than after primary suture, the changes of the peripheral end necessary to regeneration of the nerve being advanced at the time of suture. I have seen it as early as the thirtieth day. But much variability obtains and, speaking generally, the time necessary for the completion of the first stage is always long, and the interval between the second and third nearly double as long. I have never yet seen perfect sensory recovery after secondary suture, although I have

watched patients for over seven years and examined them up to fifteen years after suture. In all some difference could be appreciated between the two limbs, an area of changed sensibility remaining with imperfect appreciation of the compass test. Much less variation occurs with regard to motor recovery but the time required is invariably longer.

Nerve Bridging.—Under this general term may be included all those procedures undertaken to restore anatomical continuity when ends cannot be brought into apposition.

We have a choice of methods that have been used more or less successfully from time to time. Those of proved value may be put into four groups.

1. Transference of a portion of nerve from another source (nerve transplantation).
2. Provision of a path along which the nerve may regenerate (tubular suture, flap operations, etc.).
3. Utilization of neighboring nerve (anastomosis or crossing).
4. Shortening the limb by resection of bone.

The nomenclature at present in general use requires simplification. The same terms have been used by different surgeons to denote dissimilar operations, with results which are bewildering. I have suggested and use the following: *Nerve transplantation*; *nerve anastomosis*; *nerve crossing*. I consider that the term "nerve grafting" should be discarded: it has always meant to the English-speaking surgeon the insertion of a portion of nerve between the ends of a divided nerve, but with the recent extension of the field of operations on the peripheral nerves, it has been employed to denote also the anastomosis of one nerve to another, a meaning it conveys to continental surgeons.

Nerve transplantation is the operation of choice. Merzbacher in his experimental work found that when the portion of nerve was taken from the same animal (autotransplantation) or one of the same species (homotransplantation) degeneration occurred, but when from an animal of another species (heterotransplantation) necrobiosis and death. Forssmann found in rabbits that when the portion of nerve was taken from another rabbit, regeneration took place as rapidly as after suture, but when, from another animal, delay occurred and in many cases no regeneration. This is in accordance with clinical experience. In 1906 I investigated the recorded cases of nerve transplantation, added fresh cases, and brought others up to date. Among the 30 cases, 8 were examples of transplantation of human nerve; of these only three were reported at a sufficient interval after operation to admit of recovery. This was complete in 2. Out of 22 instances of heterotransplantation, 16 were reported at a period after operation that would have admitted of some recovery; of these only one or at most two recovered completely.

It is evident that the nerve must be obtained from the patient or from a recently amputated limb. The former is preferable and is the one that is most often employed. The upper two-thirds of the radial nerve or the internal saphenous may be used. The portion of nerve

must be removed and handled with the utmost gentleness as we wish it to play an active part. It should be of sufficient length to lie between the two ends without tension and the whole should be surrounded by cargile membrane. When it is necessary to bridge a defect in the great sciatic, a portion of nerve from an amputated limb should be used.

This method may be impossible for anatomical reasons, for example, in certain cases of division of the facial nerve in the middle ear; if the distance to be bridged exceeds four inches it is unlikely that transplantation will succeed, nerve anastomosis offers the best hope of success. In cases in which neither nerve transplantation nor anastomosis is feasible tubular suture should be performed.

Flap operations have been recommended since the days of Létievant and successful cases have been recorded, but this method leaves a complicated wound and should rarely be employed. Kenneth A. J. Mackenzie has recorded a case of resection of ten and three-quarter inches of the great sciatic nerve, in which the gap was bridged over by turning up flaps from the internal and external popliteal nerve. If methods of this kind are employed, it would be better in my opinion to entirely remove the "flap" and suture it into the gap.

Tubular suture aims at providing a path for the new axis-cylinders, free from fibrous tissue. Many substances have been used for this purpose, among them decalcified bone, collodion, preserved animals' artery. I prefer a tube composed of a portion of one of the patient's superficial veins. In performing this operation the nerve is prepared and both ends freshened, a portion of superficial vein of appropriate size is then excised and slipped over one end of the nerve. The ends are loosely united with catgut, the vein drawn over the junction, and the whole surrounded with cargile membrane. The results given by this operation are superior to those obtained from heterotransplantation.

The method of shortening the limb by removal of bone originally recommended by Löbker in 1884, and independently by Allis seven years later, has been successfully carried out by Keen and others in cases of division of the musculospiral nerve complicating fractures of the humerus. I consider it is justifiable only when, in addition to the nerve injury, there is non-union of the fracture.

The possibility of utilizing neighboring nerves attracted the attention of investigators at an early date. Two distinct operations are included—nerve crossing and nerve anastomosis.

In nerve anastomosis an attempt is made to bring the axis-cylinders of the affected nerve into end-to-end contact with some of those of the sound nerve; in nerve crossing the peripheral end of the affected nerve is united end-to-end with the central portion of a divided sound nerve. This latter operation was first carried out on animals, but surgery led the way for experimental research into the question of nerve anastomosis.

Flourens and Rawa were pioneer workers. It is, however, by the well-known work of Kennedy that its surgical potentialities were demonstrated. More recently Langley and Anderson have shown its

possibilities and limitations. Létievant appears to have been the first to recommend this method under the name of "greffe nerveuse." To Despres is due the credit of first performing the operation; he inserted the peripheral end of the median nerve between the separated fibers of the ulnar. The case was reported two months after the operation, and has been considered on insufficient evidence to be a success. It was not until 1897 that a recovery by this method was recorded. Two months after rupture of the musculospiral nerve in a compound fracture of the humerus, a flap of the median nerve was raised, and the musculospiral united end to end with it. Two years and three months later all the muscles of the forearm, except the extensor longus pollicis, acted normally and reacted to stimulation with the interrupted current (Sick and Sanger).

Excluding the cases in which the operation was performed on the facial nerve I collected 25 examples, of these 12 were reported at a sufficient time after operation to enable an opinion to be given as to the result. Two were undoubtedly perfectly successful. Four were certainly improved by the operation, and were probably successes, but the records are too scanty to enable a definite opinion to be given. Thus out of the 12 cases reported sufficiently long after the operation, only 2 were failures; some improvement took place in all the others. This is a better result than that given by Powers, who considered that 50 per cent. were successful.

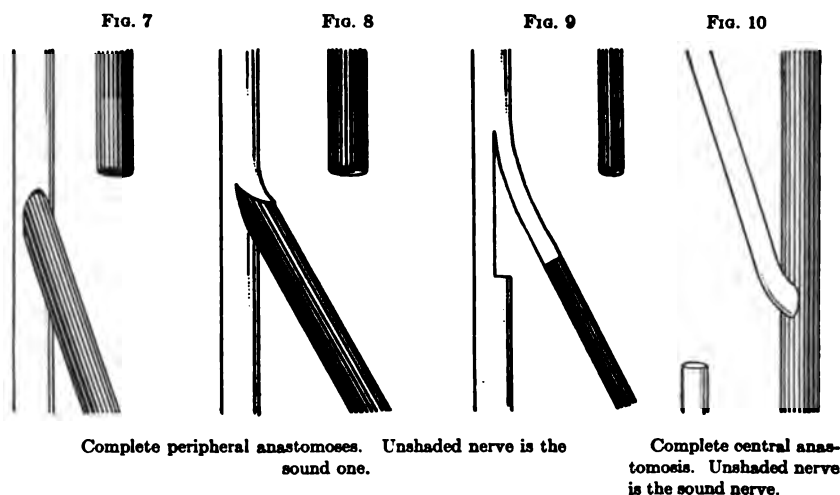
The work recently done by Kilvington is a valuable contribution to our knowledge of this subject. In his researches on dogs he found that when the central end of the divided sciatic was sutured to the peripheral ends of the external and internal popliteal nerves, recovery ensued and coördinated movements were reestablished. On counting the axis-cylinders, it was evident that a single axis-cylinder in the central end had established communication with an axis-cylinder in each peripheral end; this is important in considering what is the best operation to adopt. In his most successful case he divided the internal popliteal nerve into two portions, the smaller of which he united end-to-end with the divided external popliteal, the larger to the peripheral end of the internal, and obtained a perfect recovery in one hundred and forty-one days.

Many methods of nerve anastomosis are possible; they may be divided into the peripheral and central. In peripheral anastomosis the peripheral end of the affected nerve is brought to the unaffected nerve (see Figs. 7, 8, and 9). In central anastomosis the unaffected nerve is divided partially or completely, and its central end united to the affected nerve (see Fig. 10).

In all cases it should be the aim of the surgeon to bring the cut end of the axis-cylinders in contact, for without this, union with the central nervous system is impossible. This contact may be brought about in three ways (see Figs. 7, 8, and 9). In the case of small nerves, by making a vertical slit in the nerve a sufficient number of axis-cylinders are divided to insure a good result; but in larger nerves, a transverse

or oblique cut should be made and the affected nerve sutured in or a flap raised and end-to-end suture performed. This last method is theoretically the best, as it avoids the possibility of the union of single axis-cylinders in the central end, with one in each peripheral end.

When employed in cases of nerve injury in the limbs, peripheral anastomosis will be the operation of necessity.



Complications Arising during Recovery after Complete Division and Suture.—Pain is, as a rule, negligible; it may be present during the first few days as the result of irritation of the central end, but is rarely severe enough to need treatment.

If pain is marked it usually indicates an infective neuritis; the wound should be opened up, the junction inspected and, if necessary, sutures removed and the wound drained. In cases of this nature the wound must be again explored at a later date.

When suppuration has occurred deterioration in function may take place after a period of improvement due to involvement of the junction in fibrous tissue. This is especially liable to arise if non-absorbable material has been used for suture or the junction has not been wrapped.

Trophic ulcers may occur from the breaking down of blisters. These are due to slight injuries which pass unnoticed and do not affect sound parts. The ulcers readily heal with rest and appropriate treatment.

At the commencement of the first stage of recovery, complaint is sometimes made of pain shooting into the affected part, and at this time spontaneous blisters may originate. All tendency, however, to this ceases with the termination of this stage of recovery.

Complications Arising during Recovery after Incomplete Division.—As a rule recovery steadily advances and becomes perfect. Occasionally as the result of involvement in scar tissue the condition remains

stationary or may even get worse. Under these circumstances exploration should be undertaken.

Pain is a much more frequent complication than after complete division and is seen more often in its most severe form complicating an incomplete anatomical division when it may be associated with "glossy skin."

Occurring as the result of subcutaneous injuries, it usually arises from a direct blow. I have described several cases occurring after blows over the external condyle of the humerus, the posterior division of the external cutaneous nerve being affected. Whether the result of open wounds or subcutaneous injuries the symptoms resemble one another. Typical of these cases is the well-marked latent period, varying from one to three weeks between the injury and the onset of pain. The pain is usually described as burning or bursting in character, and is felt over the "full" protopathic distribution of the nerve, a larger area than becomes insensitive to prick on section.

It is accompanied by tenderness (hyperalgesia) easily demonstrated by lightly dragging a pin from sound to affected portions of the part. In the most severe cases, particularly those following gunshot wounds, glossy skin may arise, the typical causalgia. In these cases the pain is extremely severe and is aggravated by all external stimuli.

The degree of interference with the functions of the nerve varies, but is always incomplete.

The part should be kept at rest on a splint; this may suffice for the mild cases. This may be combined with gentle massage, electrical baths, and ionic medication. Even in the least severe cases recovery is extremely slow. If the condition is well marked, treatment of this sort is futile. The nerve must be exposed, the damaged portion removed, and anatomical continuity restored, attention being paid to the various points in operation already mentioned.

INVOLVEMENT OF TERMINAL BRANCHES

Symptoms.—Complete division of a peripheral nerve with irritation of its central end will cause pain referred to its distribution. In a similar way, irritation of a terminal branch will cause pain which may be referred to the distribution of the nerve roots from which it obtains fibers.

As the result of disease it is a familiar occurrence in the territory of the fifth cranial nerve, and the subject has become well known as the result of the work of Henry Head. It is not so well recognized, however, that irritation of one of the terminal branches of a nerve, the result of an injury, may give rise to widespread pain and tenderness. These symptoms following the wounds of small nerves were known to the older surgeons as a consequence of venesection. Abernethy, in a paper in 1793, entitled "An Essay on the Ill Consequences of Wounding a Nerve," referred to two cases. Wardrop, in 1823, recorded a case

following a wound of the thumb, and Swan, eleven years later recorded several. Hamilton, in 1838, wrote a paper on the subject, and the late Sir William Mitchell Banks in 1869.

As occurs when the trunk of a nerve is involved an interval always elapses between the injury and the first symptom. The pain is usually widespread, extending over the full protopathic distribution of the root or roots affected, and is often accompanied by hyperalgesia. There may be, in addition, particularly in the lower limb, paresis or paralysis of the muscles supplied by the corresponding nerve or root.

Cases such as these are seen at the present time most often after amputation of the fingers. It rarely happens now that symptoms due to nerve involvement follow a major amputation, for it has become one of the recognized steps of the operation to pull down and cut short the nerve trunks, but this is often neglected in amputation of the fingers.

Pain is usually noticed a week or ten days later. It involves first the stump which becomes reddish-blue, shiny, and tender; the pain then spreads to the distribution of the roots irritated, and is usually accompanied by hyperalgesia and often by redness. If neglected, the pain may spread to other nerves and later traumatic neurasthenia may develop.

Treatment.—This should be preventive. I have been struck with the number of cases in which suppuration took place. The first-aid treatment should be to surround the part with a compress wet with 1 in 20 carbolic while preparations are being made to thoroughly clean up the surrounding skin or paint it with a 2 per cent. solution of iodine in rectified spirit. While nerve involvement may take place in wounds that heal by first intention, the most severe cases are those in which prolonged suppuration occurred.

In several cases upon which I have operated, the digital nerves have been spread out in a fan-shaped manner over the end of the bone; this with the appearance of the bone led me to think that it had been divided with bone forceps before the soft parts had been sufficiently freed. If bone forceps are used, it must not be until the bone is bare. In every amputation the nerves should be seen and cut short.

When pain and tenderness develop, treatment should not be delayed. The prognosis is good if operation is performed when the pain and tenderness are still confined to the scar or stump; when they have spread to other nerves, or associated with hysteria or muscular affections, it is by no means favorable, and may, when spread has taken place, necessitate a serious operation—division of posterior roots—before relief can be obtained. Operation should be carried out with a definite object in view, to find and cut short the involved nerves. Reamputation should not be done in a haphazard way. I have notes of one patient who had had four reamputations performed without success, and now has all the complications that can result in this condition.

Sometimes it is possible to feel swellings on the ends of the involved nerves; this may be a help in finding them. In most cases it is best

to turn back the old flaps and search for the nerves at the end of the bone. If they cannot be found here, they should be sought at the sides of the finger, traced to their involvement, and cut short. It is only necessary to remove bone when the flaps will not come together again without tension. After closure of the wound the part should be kept at rest until healing has taken place. In few cases will this fail to relieve the pain. If the pain is not relieved or recurs, no further local operation should be done if the first was thorough. Before proceeding to the only certain treatment in these cases—division of posterior roots—Weir Mitchell treatment must be tried after removal of all cause for worry. It is useless treating advanced cases while compensation proceedings are pending. Weir Mitchell treatment must also be adopted after operation in the cases complicated by hysteria. When muscular symptoms are present, the affected muscles must be kept relaxed by suitable apparatus, and daily massage given until voluntary power returns. It is often twelve months before muscular recovery is complete.

Where no amputation has been performed but the symptoms result from involvement in a scar, this must be excised, the affected branch being dissected out. In a few cases amputation may be necessary.

Traumatic or "Stump" Neuromas.—When a nerve is completely divided the fibers of the upper end spread out in a brush-like manner. This "mop-like protuberance formed immediately a nerve trunk is divided," was described by Ballance and Purves Stewart as "the primitive end bulb." New axis-cylinders are developed in this, and the bulb eventually becomes a mass of fibrous tissue with small nerve fibers interlacing in all directions.

After all amputations such bulbs must be formed on the central ends of the severed nerves, but only in a few instances does their presence give rise to symptoms.

When the ends of the nerve are pulled down, cut short, and crushed with a pair of Spencer Wells' forceps at the time of the operation, symptoms rarely ensue. They arise from irritation of the bulb by direct pressure or by the traction of muscles or adhesions. The size of the bulb varies widely, and it is possible as suggested by Alexis Thomson that the increased size in some cases is due to inflammation, and that the condition has become less frequent now that the principles of Listerian surgery are carried out.

The symptoms resemble those described as due to irritation of the terminal branch of a nerve, modified by the absence of a part of the limb. There may be pain widespread in the distribution of the nerve involved, accompanied by tenderness of that portion of the stump supplied by branches from the roots involved, and in some cases by changes in the skin. The pain may be produced by direct pressure on the bulb, and is often felt with changes of the weather. Muscular twitchings often occur in association with the pain, and hysterical symptoms may be present.

The time after the amputation at which the symptoms first appear is

variable, but the longest interval that has come under my notice was twenty-three years.

The treatment should be preventive—in every amputation the nerves should be pulled down and cut short with scissors or, as suggested by Cushing, their ends anastomosed. It has been proved experimentally that crushing the end of a nerve prevents the formation of a large end bulb.

When symptoms are present the bulb and three or four inches of the affected nerve must be removed. This has sometimes failed to relieve the pain; in these cases intradural division of the posterior roots affected should be performed if the symptoms are severe.

Division of Posterior Roots.—Division of posterior roots for the relief of pain was first carried out by Sir William Bennett in December, 1888, one week before its performance by Abbé, to whom it was suggested by Dana four months previously.

The operation has recently come into prominence on account of its application to the treatment of the gastric crises of tabes and spasmodic affections (Förster).

This operation will only be called for when peripheral operations have failed. The case must be carefully studied and care taken to divide one root above and below those supplying the painful area.

The laminæ over the region to be operated on are removed, the dura is opened, and the roots divided with sharp scissors close to the cord and at their exit from the dura. In many cases it can be carried out by removing the halves of the laminæ on the affected side (unilateral laminectomy, Taylor).

Cases have been recorded by Hey Groves in which this failed to relieve the pain.

Facial Nerve.—Facial paralysis may result from injury to the nerve (a) above the geniculate ganglion, (b) between the ganglion and the point at which the chorda tympani is given off and (c) below this point. When injured at (b) taste is affected over the anterior two-thirds of the corresponding side of the tongue.

Apart from the so-called “rheumatic” affections of the nerve (Bell’s palsy), interference with the function of the nerve in the middle ear as the result of disease, or operation, is the usual cause. The nerve may suffer in fractures of the base of the skull primarily, or more often from involvement in callus. Outside the skull it may be injured during operations in the parotid region or in the removal of tuberculous glands, or from forceps pressure during childbirth; in most of these cases the “division” is incomplete and spontaneous recovery ensues.

In cases of incomplete division the usual treatment directed toward maintaining the nutrition of the paralyzed muscles must be adopted. When due to middle-ear disease it is an indication for the complete mastoid operation.

When the reaction of degeneration is present, showing that complete division, either anatomical or physiological, has occurred, the treatment to be adopted depends on the cause. If it follows a radical

mastoid operation, the sooner operation is carried out after the wound has healed the better; in cases of Bell's palsy it is justifiable to wait for six months. If the nerve is divided during the course of mastoid operation, the ends should be adjusted in the aqueduct, when restoration of function may be expected unless neuritis is set up as the result of sepsis. If discovered after the operation the electrical reactions should be tested at the end of a fortnight; if the reaction of degeneration is present, the wound should be opened up and an attempt made to adjust the ends. If this fails, nerve anastomosis must be undertaken. It must be remembered that the injury during mastoid operation is in most cases incomplete, and that spontaneous recovery follows the usual non-operative treatment.

In many cases of facial paralysis submitted to operation end-to-end union is out of the question, and a neighboring nerve must be utilized. This was first carried out by Drobnik in 1879, who performed nerve crossing between the peripheral end of the facial and the central end of the spinal accessory. The modern operation is, however, due to the work of the Ballances and Purves Stewart. The nerves that have been used are the spinal accessory or one of its branches, and the hypoglossal; they have been completely divided, and end-to-end union performed with the peripheral end of the facial (nerve crossing), or united to the side of the nerve (anastomosis). In 1895 C. A. Ballance anastomosed the facial to the spinal accessory. Körte first suggested the use of the hypoglossal, and this nerve has been used with success by Ballance, Taylor, and Clark among others. The hypoglossal is the nerve of choice; dissociated voluntary movement is restored more quickly after anastomosis to this nerve than when the spinal accessory is used. Nerve anastomosis, and not nerve crossing, should be performed; there is no necessity to sacrifice the hypoglossal nerve: emotional dissociated movement will return without.

In order to carry out the operation, a long incision should be made extending from the mastoid at the level of the external auditory meatus down to the great cornu of the hyoid. The anterior border of the sternomastoid is pulled back, and the posterior belly of the digastric identified after separation of the parotid. The digastric is then pulled backward, and if large it may be necessary to divide its upper border. The facial nerve is identified, this being most easily done by feeling for the styloid process of the mastoid; the nerve passes out immediately in front of this and enters the parotid gland. The nerve is freed, and an attempt is made to pull the stump out of the foramen in cases in which there is reason to believe that the nerve was divided in the course of an operation on the middle ear. If this cannot be done the nerve is divided in the foramen as high as possible with a tenotomy knife. The hypoglossal must next be sought; the transverse process of the axis and the occipital artery are useful guides. The internal jugular vein is next found and retracted inward; this exposes the hypoglossal nerve. With it is running the vagus, but the hypoglossal is easily identified from the course it is taking. It is freed and brought

toward the facial; a flap consisting of about one-third of its fibers should be carefully raised and the peripheral end of the facial united end-to-end with it by fine catgut. The raw surface and the junction should be surrounded with cargile membrane.

The after-treatment must be patiently carried out. The nutrition of the muscles must be kept up by massage and the application of a constant current until such time as voluntary power begins to be restored. As soon as voluntary movement returns to each group of muscles they must be exercised systematically until the patient regains complete control.

The prognosis varies with the cause of the paralysis, being better when the division results from injury than when it is the result of neuritis. Suppuration of the operation wound renders success doubtful. The first sign of recovery usually appears about the third or fourth month, the face while at rest becoming more symmetrical, although there is no return of voluntary power. A few weeks later it is noticed that the angle of the mouth can be moved, at first only with movements of the tongue; then the muscles of the upper lip, and finally those of the forehead. With exercise, the movements become dissociated, and finally emotional movement may return.

For a few weeks after operation the side of the tongue is paralyzed, causing difficulty in speech and deglutition; this passes off but the affected side of the tongue may remain smaller for a considerable time.

In 40 cases collected by the writer, improvement occurred in all reported at a sufficiently late date; but in comparatively few did emotional movement return, the face on the side of the injury remaining immobile in smiling. It is safe to say that in the majority of cases the appearance of the face at rest will become normal.

Vagus Nerve.—The trunk of this nerve, although exposed to many forms of injury, rarely suffers. Its recurrent laryngeal branch suffers most often in operations upon the thyroid, but is rarely completely divided. Schloffer reported that he had seen an affection of the recurrent laryngeal nerve thirteen times in 505 operations, but that it was usually of a transitory nature. Leischner confirmed this. Occasionally, however, the nerve is completely divided and spontaneous recovery does not ensue.

The nerve appears to have been first sutured by Föderl in a case in which the trachea was divided, but the first deliberate operation on the nerve was undertaken by Shelton Horsley two months after division in a bullet wound; fifteen months later recovery was almost perfect.

When laryngeal paralysis results from operation, at least three months should be allowed to elapse before exploration is undertaken. If no recovery has taken place, operation should be undertaken on the usual lines.

Spinal Accessory Nerve.—The external or spinal portion of this nerve is not infrequently divided during the course of operation upon the neck, particularly during the removal of tuberculous glands. In

many of these cases the branches of the cervical nerves to the trapezius are affected at the same time producing its complete paralysis. The extent of supply of the spinal accessory and the cervical nerves to the trapezius varies; as a rule the upper trapezius is paralyzed by division of the spinal accessory alone.

If the nerve be divided during the course of a surgical operation it should be immediately sutured. The deformity resulting from paralysis of the trapezius is marked, and an attempt should be made in every case to deal with it by operation. It should be exposed through an incision along the anterior border of the sternomastoid, and an attempt made to perform secondary suture. If it be impossible to bring the ends into contact or to find the upper end, the peripheral end should be anastomosed to the anterior primary division of the third and fourth cervical nerves. When the cervical branches are divided in addition, an attempt must be made to obtain the nerve supply again from the third and fourth cervical by suture or anastomosis.

If it is found impossible to unite the severed nerves, muscle transplantation should be carried out. A portion of the latissimus dorsi detached from its insertion into the humerus may be fixed to the dorsum of the scapula to take the place of the lower trapezius. Flaps from the middle and upper part of the sound trapezius may be turned over to take the place of the paralyzed upper part. This was carried out in a case recorded by Katzenstein.

The Brachial Plexus.—The plexus may suffer injury (1) above or (2) below the clavicle; in the former case it is usually the anterior primary divisions of the cervical nerves entering the plexus that suffer; in the latter, its cords. Most of the injuries are subcutaneous; above the clavicle, the result of traction; below, the result of pressure. Penetrating wounds are rare in civil practice.

Supraclavicular injuries are usually the result of indirect violence, the force being applied to the head or shoulder, but they occasionally arise from the presence of a cervical rib, or the result of a penetrating wound. As the actual lesion produced and the prognosis differ each must be discussed separately.

The lesion in these cases is the result of overstretching. This was first demonstrated by Sir Victor Horsley as the result of experiments made in 1884. Clark, Taylor, and Prout have shown that brachial birth paralysis owns the same cause. The immediate lesion consists in a tearing of the perineural sheath with complete or partial severance of nerve fibers at different levels.

When the violence is applied to the head or shoulder the traction falls first upon the upper part of the fifth anterior primary division, then upon its junction with six, following this upon the remaining divisions in order from above downward.

But if the traction fall upon the plexus from below—for example, a man in falling from a height endeavors to save himself by clutching at some projection—the injury affects the first dorsal, then the others in order from below upward. The same may result in infants in

breech presentations, with the arms extended, or in certain face presentations.

In both cases recovery takes place from the root last affected, and may leave Erb-Duchenne or Klumpke paralysis as a terminal lesion when the original affection was more widespread.

Infraclavicular Injuries.—In the majority of cases these are the result of pressure, most often from the head of a dislocated humerus, occasionally from reduction of the dislocation by the "heel in axilla" method. In other cases they may be due to fracture of the humerus or of the neck of the scapula.

The whole plexus may be injured. The result on sensibility is in most cases the same as that seen in supraclavicular, but when due to direct violence applied from without, the lesser internal cutaneous nerve also is involved, and the sensitive area on the inner side of the arm is not present. Pupillary symptoms are absent, and the serratus magnus muscle often escapes.

The inner cord of the plexus is most often affected, then the posterior, and finally the outer.

Diagnosis.—In every case of brachial plexus lesion, in addition to the fact of complete or incomplete division, the exact point of injury must be ascertained.

There are four important symptoms: (1) The condition of the scapular muscles. If the serratus magnus is unaffected, the lesion is not of the anterior primary divisions; it is sometimes possible to locate an injury to between the points at which the nerves supplying the spinati muscles and the serratus magnus are given off. (2) The grouping of the paralyzed muscles; for instance, it is obvious that no injury below the clavicle will paralyze the Erb-Duchenne group. (3) The condition of the pupil. (4) The presence or absence of anesthesia.

After diagnosis of the exact spot, treatment must be carried out along the lines already laid down; complete injuries must be explored and the appropriate treatment adopted. When the whole plexus is affected, as the result of indirect violence, *e. g.*, falls on the shoulder, exploration should be undertaken immediately, without waiting to ascertain whether the division is complete or incomplete. In no instance in which operation was undertaken at a late period after the accident has the result been satisfactory. Incomplete division of the fifth anterior primary division or upper trunk may result in the development of the reaction of degeneration in the deltoid and spinati. This should always be treated by operation, and the damaged portion, which will be found on the upper and outer aspect of the nerve, resected and a portion of the radial nerve transplanted; or this damaged portion may be anastomosed to the anterior primary division of the sixth cervical nerve.

Prognosis.—It is well established that injuries of the brachial plexus have a worse prognosis than those of the peripheral nerves.

V. Bruns found that while spontaneous recovery ensued in 66 per cent. of subcutaneous injuries of peripheral nerves, only 26 per cent.

of similar plexus injuries got well spontaneously. Warrington and Jones from the examination of cases under their care found spontaneous recovery in 30 to 40 per cent. These unfavorable figures are due in part to the nature of the injury. In a large proportion of cases the nerves are overstretched, and this results in hemorrhage into the sheath and consequent fibrosis; in addition, if it leads to rupture, the fibers give way at different levels; hence spontaneous recovery is unusual when the signs of complete division are present, and is apt to be imperfect in cases of incomplete division. Again, it is possible that the injury in some cases tears the roots away from the cord. Even after operation the prognosis is not so good as, for example, after secondary suture of the median at the wrist or the musculospiral. This has to do to a great extent with the length of time necessary to complete recovery; in many cases the patient ceases to attend for efficient after-treatment, and when recovery of the nerve has finally become complete, the muscles are atrophic, and contractures of the opponent muscle render the regeneration of the nerve futile. Careful treatment at the time of the injury, and unceasing, patient after-treatment, will improve the prognosis. It cannot be too strongly impressed upon the patient that the operation only puts him in a condition favorable to recovery, and that possibly years of patient treatment must be carried out if success is to be obtained.

Brachial Birth Paralysis.—As just mentioned, these usually result from traction, but it is possible that some of the milder cases are the direct result of pressure.

If seen early the arm should be bandaged to the side over cotton-wool with the paralyzed muscles relaxed and kept at rest until tenderness has disappeared when massage and movements should be employed, particular care being taken in the Erb-Duchenne type to prevent the development of a forward rotation of the shoulder. The electrical reactions of the affected muscles should be tested at the end of three months; if the reaction of degeneration is present operation should be performed so soon as the condition of the child will permit. Of the cases under my care 65 per cent. have recovered completely without operative interference.

Operation appears to have been first carried out by Kennedy in 1900, and it is in great measure owing to his work and the exhaustive researches of Clark, Taylor, and Prout that the treatment of this condition has been established on a sound basis.

The upper cords of the plexus may be exposed through an incision in the posterior triangle of the neck. The anterior primary divisions of five and six must be freed, together with their junction and division, and the origin of the suprascapular nerve. The scar will usually be found in five just at its junction with six; in some cases the upper trunk will be found damaged. In other cases the fifth anterior primary division is found torn through and occasionally the suprascapular nerve also. The damaged portion of the nerve must be removed and the ends united in the usual way. No difficulty will be experienced

in most cases in bringing the ends together. If there be tension, the shoulder should be elevated and the head inclined to the affected side. If the ends cannot be brought into apposition, or the upper end cannot be found, nerve anastomosis should be carried out. If the lesion involves the lower divisions, it will be necessary in most cases temporarily to divide the clavicle.

Postanesthetic Paralysis.—Under the term postanesthetic paralysis many nerve injuries of the upper limb are included. Most are of the Erb-Duchenne type and so, obviously, supraclavicular; others from their distribution are undoubtedly infraclavicular, and in a few direct pressure has affected individual nerves, usually the musculospiral.

These injuries of the plexus are by no means uncommon, although published cases are few. Cotton and Allen in 1903 were only able to collect thirty from the literature. It occurs in patients in whom during the course of the operation the arms are abducted and externally rotated or raised above the head. The right arm is usually affected. Stretching over the head of the humerus with the arms elevated above the head is the probable cause of the infraclavicular injuries. In all cases the lesion is incomplete, corresponding to the slight violence which produced it.

The prognosis is good: all the cases that I have had under observation have recovered completely without surgical intervention, and all except one of those collected by Cotton and Allen. The treatment should be preventive; such cases occurring as the result of a routine abdominal operation are a reproach to all concerned.

Injuries Due to the Presence of a Cervical Rib.—When symptoms result from the presence of cervical ribs they are in 70 per cent. of the cases nervous in nature. They occur more often in women, and on the right side. Of 10 cases under the care of the writer all were women; the symptoms were on the right side in all except 2, and of a nervous nature in 9.

Our appreciation of this condition dates from Thorburn's paper in 1905. Although a case had been operated upon by Cooté many years before, the condition appears to have escaped notice. Lewis Jones in 1893 drew attention to atrophy of the thenar muscles in young people, and on investigating the cases thirteen years later found that 10 out of 14 were the possessors of cervical ribs. Important papers bearing on treatment have since been published by Keen and by Hinds Howell.

Symptoms usually appear in early adult life, and are due in most cases to pressure upon the lowest trunk or nerve entering into the plexus. In many cases they consist of wasting of the intrinsic muscles of the hand, most marked and starting in those of the thenar eminence; if of long standing the reaction of degeneration may supervene. In other cases the principal complaint is pain down the ulnar border of the forearm and hand, or a general weakness or heaviness of the whole limb noticed at the end of the day. Sensory changes are unusual and when present rarely exceed epicritic loss.

The condition should be thought of in all cases of "brachial neuralgia," or of wasting of the thenar muscles. The ribs can usually be felt but occasionally can only be demonstrated by *x-ray* examination.

Although a cervical rib is present it does not necessarily follow that it is the cause of the symptoms. Several cases have come under my observation in which a cervical rib was removed from a patient suffering from syringomyelia to which the symptoms were due. Thomas Murphy has recorded a case in which the symptoms were due to the pressure of the first rib. A similar case has recently been under my care.

Treatment consists in removal of the rib together with the periosteum covering it, followed by careful after-treatment.

The rib is most easily exposed through a vertical incision behind the sternomastoid, remembering that it arises just below the level of the cricoid cartilage. After pulling forward this muscle it should be reached by blunt dissection, its central end disarticulated if possible and cleanly removed. Care must be taken to avoid injury to the plexus and the pleura.

The Long Thoracic Nerve (Nerve of Bell).—This nerve is most often injured in males between the ages of twenty-five and forty, usually on the right side. Generally caused by prolonged pressure in the supraclavicular region, it occasionally follows violent muscular efforts and direct violence applied to the shoulder.

Paralysis of the serratus magnus rarely occurs alone, it is usually combined with paralysis of the lower trapezius.

If seen early, avoidance of all injurious pressure and absolute rest to the arm should be ordered, followed by massage and stimulation with the interrupted current applied to the muscle. If the paralysis persists, and the reaction of degeneration develops nerve anastomosis should be performed, or the insertion of the sternocostal part of the pectoralis major may be transplanted to the lower angle of the scapula. It is usually impossible to deal directly with the damaged portion of the nerve or the branches of which it is composed.

The Circumflex Nerve.—This nerve is liable to injury where it turns round the neck of the humerus as the result of subglenoid dislocations, fracture of the surgical neck of the humerus, or neck of the scapula; by direct violence, as in a case recorded by Robert Kennedy, or in sleep, crutch, and anesthetic palsies. It may also suffer injury in miners who lie for long periods on their left side, and be secondarily involved in inflammatory processes from the shoulder-joint or the subdeltoid bursa.

Injury to the circumflex nerve is by no means so common as is usually supposed. It has been said to follow direct blows on the point of the shoulder, but in most cases the injury is to the anterior primary division of the fifth cervical nerve, and careful examination will show that the spinati also are affected. In other cases the wasting of the deltoid on which the circumflex injury was diagnosticated is found to be, in common with all the muscles around the joint, the result of a traumatic arthritis.

Careful examination is necessary before coming to a decision with regard to treatment; testing must be carried out for all forms of sensibility. If there is no loss of sensibility, and there is paralysis of the deltoid with the reaction of degeneration, it is extremely improbable that the circumflex nerve is injured. If the signs are those of complete section of the nerve, the age of the patient and his occupation must be taken into consideration; in some cases operation can be avoided by training the neighboring muscles to take the place of the deltoid. This vicarious restoration is well illustrated in a case recorded by Kennedy. If it is essential that perfect movement be obtained, operation must be carried out. The nerve should be exposed in the axilla just before it passes through the quadrilateral space and traced onward.

Ulnar Nerve.—This nerve may be injured at the elbow or at the wrist above or below the point at which the dorsal branch is given off. While it may suffer in any part of its course from injury and the result of penetrating wounds, these are more usual at the wrist. At the elbow the injury is due in most cases to fracture or deformity, recent or old, usually the latter, and to dislocation of the nerve.

If complete recovery is to obtain the paralyzed muscles must be kept relaxed. I have devised an aluminum splint for this purpose worn on the dorsum of the hand, keeping the fingers slightly flexed at the metacarpophalangeal and extended at the interphalangeal joints. If this is worn the onset of "claw hand" may be prevented and function be completely restored.

Injury the Result of Deformity at the Elbow.—Injury in connection with recent fracture is not uncommon. Bruns records that in 101 fractures of the humerus the ulnar nerve suffered in 14. As a complication of a dislocation of the elbow-joint it is rare.

The division is usually incomplete physiological, and recovery is rapid.

More important are the lesions resulting from the deformity, and first making their appearance after a lapse of several years.

Attention was first directed to the subject by Panas in 1877. He recorded 4 cases, of which three belonged to this group, and mentioned that the condition had led to errors in diagnosis and that Duchenne had at first mistaken a case of this description, "in common with other eminent medical men," but that careful attention to symptoms had enabled him to make the correct diagnosis.

These observations seemed to have escaped general attention, for ten years elapsed before any further cases were recorded; Weber in a paper upon the etiology of paralysis of the ulnar and median nerves from peripheral causes, described 2 cases, in 1 the injury dated back twenty-seven years, in the other thirteen years. In 1908 I collected all the published cases and added two of my own. Since then I have had 14 cases under my care.

The injury producing the deformity has been, in most cases, a fracture or epiphyseal separation of the lower end of the humerus which

has led to a marked cubitus valgus, often with obliteration of the ulnar groove. The nerve is enlarged into a spindle-shaped swelling behind the internal condyle.

The symptoms appear usually many years after the injury. In most of the recorded instances the accident was in childhood, and in several cases no history of the injury could be obtained. This was the case in one of the patients upon whom I operated, but the physical signs and the radiograph left no doubt as to the origin of the marked cubitus valgus which was present.

The first symptom noticed is generally pain in the distribution of the nerve; this is soon followed by wasting and weakness in the muscles which it supplies. This may have gone on to complete division, but, as a rule, the signs are those of incomplete division only.

Diagnosis.—If the existence of this condition is remembered, no difficulty should arise. Dislocation of the ulnar nerve may be suspected, but the absence of undue mobility and the gradual onset of symptoms render differential diagnosis easy. In many of the cases the diagnosis of anterior poliomyelitis has been made. Careful attention to the symptoms will prevent this mistake. The typical deformity in the region of the elbow and the spindle-shaped swelling upon the nerve establishes the diagnosis.

Treatment.—In all cases means should be taken to remove the cause of the disease. In the least severe cases, rest will entirely relieve the symptoms for a time, but recurrence inevitably takes place on resuming active use of the forearm.

The nerve should be exposed behind the internal condyle and a groove in the bone chiselled for it; if the cubitus valgus is excessive, it may be necessary to remedy this. When the signs are those of incomplete division only, removing the cause of the pressure and protecting the nerve will be sufficient, but when the signs are those of complete division, the spindle-shaped swelling should be excised and end-to-end suture performed in addition.

Dislocation of the Ulnar Nerve.—Abnormal mobility of the ulnar nerve is of common occurrence, and gives rise to no symptoms; it is found in individuals in whom physiological cubitus valgus is more marked than usual, occasionally in those in whom cubitus valgus results from injury. This condition, which may be called subluxation of the ulnar nerve, is the predisposing cause of dislocation, which name should be reserved for those cases in which the nerve travels over the internal condyle. This can occur only in flexion of the forearm, and in most cases a fall on the flexed elbow is given as the cause. This probably ruptures the fascia which keeps the nerve in position.

The condition occurs most often in males between the ages of twenty and thirty. The symptoms usually come on immediately after the injury; occasionally some time elapses, the frequent injuries to which the nerve is subject in passing over the internal condyle causing fibrosis. In a few instances the condition originates without injury; in these patients the fascia which keeps the nerve in place is gradually stretched.

If symptoms are present pointing to interference with the functions of the nerve, treatment must not be delayed. A long incision is made to expose the ulnar nerve in its groove which is deepened after freeing the nerve; it should then be wrapped in membrane to prevent its becoming adherent, and finally the groove formed into a canal by stitching a portion of the fascia of the triceps over it. In cases that have been neglected, in which a diffuse fibrosis is present leading to all the signs of complete division, it will be necessary to resect the damaged portion of the nerve; this will, however, rarely be required.

Musculospiral Nerve.—The special interest in connection with this nerve lies in the fact of its frequent injury in association with fractures of the humerus, particularly of its lower or middle thirds. Von Bruns found it complicating 8 per cent. of 886 collected cases, Riethus 4 per cent. of 319 cases. The former figures correspond with my experience.

In addition, crutch, "sleep," and "Saturday night" paralysis are not uncommon as the result of direct pressure upon the nerve.

In every case the paralyzed muscles must be kept relaxed by suitable splints, and in no case must "wrist drop" be allowed. Operation is rarely indicated except in the cases complicating fractures, as the injury is incomplete and recovery is usually rapid.

Treatment when involving fractures is given on page 76.

The prognosis is better than after injury of any other nerve. In cases in which neurolysis is necessary, restoration of function often commences within a few weeks. Even after secondary suture, motor power usually returns within a year.

Great Sciatic Nerve.—Injuries of the great sciatic nerve are rare. It may suffer in penetrating wounds, but in civil practice usually from traction due to the reposition of a dislocation of the hip, congenital or acquired. It is important to remember that its external popliteal (peroneal) division suffers most in these injuries, and signs of its complete division may be present without affection of the internal popliteal portion.

Treatment follows the usual lines and recovery is always slow.

External Popliteal Nerve.—This nerve suffers most often as the result of direct violence as it passes around the neck of the fibula. When primarily injured in association with fracture, operation should be undertaken at once, for the nerve not infrequently passes between the two fragments.

However caused, the division is usually incomplete, but recovery is always slow and, unless great care be taken, often imperfect. It is essential to keep the foot at right angles and prevent foot drop.

External Cutaneous Nerve.—Injury to this nerve is of importance in that it is occasionally the precursor of Bernhardt's disease. The nerve is usually involved at its exit from the deep fascia and in many cases a spindle-shaped swelling may be felt here. The injury causing it may be long continued, as by a badly fitting truss or in other cases it follows a sudden strain.

If due to any continued pressure, removal of the cause is often followed by cure. When there is a definite spindle-shaped swelling, resection, as advocated first by Chipault and independently by Spiller in 1898, should be carried out and the nerve continuity reestablished. This treatment is, however, useless unless the condition is definitely due to injury.

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CHAPTER III

TREATMENT OF THE MUSCULAR ATROPHIES AND DYSTROPHIES

By I. ABRAHAMSON, M.D.

INTRODUCTION

THE progressive muscular diseases are generally regarded as being so hopeless that the neurologist, as well as the general practitioner, considers his duty fulfilled when he reaches a diagnosis. He then secretly imparts to the family the bad outlook, and, to mitigate the evil nature of his tidings, mercifully suggests that electricity and massage may be of some value, but with little faith that the measures he proposes will have any beneficial effect on the disease process.

Etiology.—Although the etiology of these conditions leaves much to be desired, still we must remember that with every advance in medical knowledge the underlying cause of another disease is discovered, and the etiologic scrap heap is steadily diminishing; yet there are a multitude of diseases in which the essential etiologic factor still evades us, so that we must be satisfied with many alleged factors, among which the true causal element may or may not be included. The relative values of the given causes must for the time being remain a matter of opinion. We speak of a principal etiologic factor, with secondary or contributory factors, and, as a rule, regard external circumstances as contributory factors. But many of the conditions to be spoken of here are not separate diseases, but merely symptom-complexes—groups with superficial resemblances in which the external physical and chemical signs of the internal morbid process are similar. The degree of outward correspondence is inadequate to constitute any real unity or identity. The same clinical entity cannot arise from two different causes. Conversely, two different exciting agents must produce two separate morbid conditions.

There are in the main two chief classes of diseases—one endogenous, due to defects in formation, development, and vitality of certain parts of the organism; the other exogenous, due to chemical, mechanical, thermal, parasitic, or other causes. Endogenous causes are hereditary in character, and can be proved not only by the logical exclusion of other factors but also by the presence of the results of these causes in more than one member of the same family. Exogenous diseases are due to a diversity of irritants. The separation of exogenous diseases from one another is easier than that of hereditary diseases where

with a unique etiologic basic factor many clinical entities exist which are differentiated only for convenience of study.

However, as we shall see in the study of myopathies, etc., absolute separation is impossible. The localization of the disease process may be inconstant, but owing to the existence of transition forms no site or sites can claim a monopoly. This variation of localization exists not only with reference to endogenous diseases, but to exogenous diseases also, *e. g.*, tuberculosis and carcinoma. In exogenous diseases a difference in site very often necessitates a difference in prognosis and treatment. In endogenous diseases localization does not profoundly modify the course. When we later study the atrophies we shall see that too much attention has been paid to clinical form and too little to underlying cause.

Heredity.—I shall not go into a detailed description of the principles governing heredity, nor of the laws of transmission. Heredity is the transmission of traits or characteristics. These may be similar, *i. e.*, direct; or dissimilar, *i. e.*, indirect. The laws of Mendel, Galton, and others governing certain types of heredity can be studied elsewhere.

Four types can be distinguished:

1. Forms with latent heredity, the parents being apparently normal, one-sided heredity like the sports of the botanists.

2. Forms with consanguinity—spastic paralyses, retinitis pigmentosa, deaf-mutism.

3. Double heredity—albinism.

4. Forms with sexual differentiation: (*a*) Females remain unaffected, but transmit the disease to their male descendants. The males do not transmit the disease. This sexual differentiation is seen in hemophilia, hemeropia, heredo-atrophy of the optic nerve, dystrophies, extra digits and toes, Merzbacher's aplasia axialis extracorticalis congenita. (*b*) Cases with the converse (Higier) familial periodic paralysis. The germinal diseases (endogenous) in some cases show destruction of entire systems; in others very limited involvement. Some observers maintain that the germ plasma changes do not markedly differ originally, but that in later life exogenous causes determine the nature and extent of the lesion. This is true to a great extent for progressive muscular atrophy, tabes, paresis, etc., but for various familial conditions it is false. Further research into the conditions that govern germinal activity is necessary to elucidate this matter.

Are the primary changes situated in the nervous system or in other organs? What part does faulty metabolism play? Does deficient nutrition or retention of deleterious substances in the blood or lymph exert the main influence? We know of the extreme sensitiveness of the nerve cell to various toxic influences. When one regards states like amaurotic idiocy, with its sudden and widespread abiosis, the importance of the exogenous factor in the blood, or lymph, seems as great or scarcely less than the endogenous factor.

Summing up: The hereditary trait consists (1) of a predisposition to certain diseases, induced by disturbances in the metabolism, by

blood diseases, by various intoxications and other constitutional aberrations, which possess in common a selective affinity for the nervous system, *i. e.*, acquired tendency; and (2) of an increased vulnerability of certain organs, due to germinal anomalies of the inherited tendency.

Whether the dynamic mechanism is an accelerated metabolism, or an augmented degeneration or regeneration, is not exactly known.

The various noxa which may be able to produce certain alterations accelerate and increase the destructive changes already existent, or which are about to be initiated as a result of the hereditary trait.

The great chronicity of the progressive muscular diseases enables the practitioner to recognize them in their early stages, and to institute measures for their retardation, if not for the prevention of their further development.

These diseases, by reason of the early stage at which they usually come under observation, ought to afford ample opportunity for therapy, but, owing to their chronicity, and the attitude of hopelessness which the physician assumes toward them, few conditions are less improved by medical supervision.

The family physician, confronted by an hereditary disease, ought not to regard it as possessed of a relentlessly predestined course. He should at once institute therapeutic measures, and he should patiently persist in his endeavors. The majority of these cases may not be amenable to treatment, yet a definite number not only cease to deteriorate, but even improve or actually advance toward cure. Even where an hereditary taint exists, not every progressive condition is due to this heredity. It may be due to causes the consequences of which are quite amenable to treatment.

Prophylaxis.—Prophylaxis demands the consideration, not only of the individual, but also of the family. Good-will and self-sacrifice are as necessary as favorable material conditions. The apparently unaffected members of the family must be carefully observed, a neuropathic taint (meaning commonly, increased irritability and exhaustibility, *i. e.*, irritable weakness) may give rise, in some to the so-called functional nervous diseases, and in others to organic diseases. There is apparently no known reason for this selection between the functional and organic disease, but it undoubtedly exists, as a perusal of family trees will show.

Neuropathic taint is, in many cases, merely a predisposition. If the various exogenous causes are removed, or protected against, much can be accomplished to offset the predisposition.

Neuropathies are not of necessity transmissible. We find families in which there may be three or more children with dystrophies, yet the antecedents are unaffected. Indeed, if transmission were certain, such families would have been long ago wiped out of existence.

How shall we combat the neuropathic taint?

Regulation of Marriage.—First, by attacking the problem at the root by the regulation of the marriage of individuals from affected families.

This is the province of eugenics, which may well be considered elsewhere. The interdiction of marriage is a dubious good. It cannot regulate the birth of illegitimates, who in foreign countries, and among our immigrant population, form a large percentage of the children born; it cannot regulate latent or indirect heredity, which is much more frequent than direct. Then again, absolutely to interdict consanguineous marriages is too drastic a remedy, because of the rarity of the hereditary conditions resulting therefrom. Lastly, a study of tainted families shows, after all, only a small percentage of cases of transmission. Interdiction would tend to immorality and increase the illegitimate birth rate. If an unaffected member of an affected family were denied the right to marry it would tend to the increase of venereal disease.

What advice should be given to a member of such a family by his physician on the subject of marriage? The latter should make a careful study of both family trees. Even in the absence of accurate tables or statistics, he should clearly explain the laws of heredity; the danger to the offspring resulting from such an union; and the agony to the parents and to the child should the hereditary disease develop.

An individual of such a tainted family might be advised to marry into one free from bad heredity. The union of similar heredities increases the morbid tendency in the offspring.

The interdiction of marriage often results in enhanced desire, and in women the instinct of self-sacrifice frequently nullifies our efforts.

The physician must weigh the various factors, and determine the course to pursue according to the individual; then, being certain of the correctness of his attitude, must firmly urge the adoption of his views. He must bear in mind that, for the individual himself, a normal marriage, with its prospects and its regularity of living, is in itself a natural barrier to the development of the neuropathies. But the religious idea that marriage is for weal or woe must be combated energetically where the woe is the more likely consequence. Should the physician be unwilling to assume the full responsibility, it would be advantageous to consult with some eminent authority on the subject. So much for the question of marriage.

Prevention of Conception.—Now similar considerations must pertain to the prevention of conception where two or more children of a family have distinctly shown signs of an hereditary disease, let us say of primary myopathy.

I am in favor of the prevention of conception in such cases. While appreciating fully how much the parents desire a normal child, the physician in view of the sad results, and their very likely repetition, ought to advise against conception.

Hygienic Measures.—What steps shall we take to do all humanly possible to prevent the occurrence of the family blight in the still normal children of a tainted family?

Hygiene must begin in infancy. We should where possible insist upon breast feeding, careful regulation of diet, the avoidance of too

early walking, and all forms of overexertion. There must be individualization in the hygienic management.

Hygiene in childhood is still more important. The principal article of diet should be milk; after dentition, a mixed diet; meat only once a day, at noon; cereals, bread, etc., in abundance; very few sweets; no coffee, tea, alcohol, spices, nor pungent sauces. The regulation of quantity is as important as the regulation of the kind of food given.

The child must have plenty of light and air; coddling must be avoided; a process of hardening, gently and gradually applied, is very useful after the first year; the child must get accustomed to noise, to changes of temperature, and to changes of weather. Cold baths, shower baths, and ocean bathing should be employed only after the fifth year.

Clothing must afford sufficient protection against the elements; a rational dress is advisable, loose porous materials permitting sufficient evaporation from the skin. Tight clothing, excessive local bundling or wrapping are to be guarded against. The neck ought to be free. The night clothing must be regulated along similar lines.

The care of the skin, by proper washing and bathing, is of prophylactic importance.

The regulation of sleep is the next factor. Up to four years of age it is best for children to have a nap by day. They must be taught very early to distinguish night from day, and hence the room ought neither to be darkened in the daytime nor artificially illuminated at night. Children up to ten years of age ought to be in bed at 8 P.M., and rise between 6 and 7 A.M.; in later years sleep from 9 P.M. to 6 A.M. is sufficient.

So much for the more elemental needs of the child.

Physical and Mental Training.—Physical and mental training ought to go hand in hand during childhood. Ziemssen says: "The glorious results of sport and play show their effects on the body and on the mind. With increase of skill and strength, there arises the consciousness and confidence in one's own powers and capacity, courage increases and aggressiveness results. As a matter of course, with the disappearance of the physical atony, there vanishes also the atony of the will; character, energy, and judgment are enhanced." The physical mollicoddle is only too often the mental one.

When we regard the progressive muscular atrophies, we see the importance of trauma as an etiological factor, and in our more complete attempts at prophylaxis we must use every effort to avoid trauma. No doubt the importance of trauma is popularly much exaggerated. Parents ascribe practically all progressive diseases of obscure causation to some trauma sustained by the child. We must know, however, that as contrasted with adults, children are not prone to severe sequelæ following injuries, and that the processes of regeneration are more active in children. Nevertheless, if lesions of bones and joints occur in childhood, greater defects can follow than in adults. But injuries are unnecessary. Sufficient safeguards must surround the child.

Children should be left to their natural development in their earliest

mental education. Precocious forcing is inadvisable. The inherent mimicry of children suffices for the normal child; cleanliness and the main moralities can be impressed. Disorderliness, gross disobedience, extreme selfishness must be combated; the emotions must be controlled and regulated. Too great leniency in this direction promotes bad temper, stubbornness, and greediness, all tending to the development of the neuropath.

The child should be unacquainted with fear, and corporal punishment should be very rare and very timely, the temporary deprivation of pleasures ought to be sufficient in most cases for the purpose of correction. Our aim must be to develop as normal a child, physically and mentally, as is possible in the given case. Later, should an atrophy or a dystrophy develop, the careful and trying immediate treatment can be carried out with the least resistance on the part of the patient, and with better hope of success.

And similarly with school work. We must not forget that, at best, it is class work. The individualization must be done at home, and home work must supplement but not add to the burdens of the school. Half sessions for the first few years of school life are advisable.

The school physician's duty is a very important one, and in time will equal in value that of the family physician. The earliest evidences of chronic disease will often be recognized in the schools by an observant teacher, nurse or physician. Aside from regarding only the child's physical powers, as is done at present, experienced medical advisers should be consulted on the arrangement of the curriculum for the healthy child, as well as for the physically and mentally unfit. The less said about the psychological knowledge of our educational authorities the better. Physicians versed in psychology as well as in physiology ought to supervise the books given to children; they should segregate the psychopathic and neuropathic children, and devise ways and means to combat morbid tendencies.

Physical and mental precocity must be mitigated; the emotions ought to be properly developed and nurtured. Dry studies must be tempered by those with pleasurable feeling tones, and higher sentiments should be encouraged. Love of truth, tolerance, and proper humility must be fostered. Laziness, idleness, and indolence must be combated. Lack of occupation tends to the development of neuropathies. Pleasure must be regulated and tempered; too much company, excesses of all kinds must be forbidden.

The choice of occupation is an important one, and in a family with a dystrophic taint the family physician ought to be consulted about the choice of work. Open-air labors, as free from danger of trauma and from overexertion as possible are preferable. Caste prejudice must be laid aside and not allowed to stand in the way of a proper choice.

The accidents of childhood must now be considered. We have already spoken of trauma, we must now consider the infectious diseases and their role in the production of neuropathies.

While the central nervous system, including the sympathetic, is

the regulator of the various animal and vegetative functions, it is conversely affected by disorders of function of the various organs; it responds very promptly to perturbation of their action, but only in rare cases is the central nervous system permanently affected by visceral disease. Nevertheless, this interrelation is of great importance; the family physician must bear it constantly in mind, and the neurologist must be in a position to recognize the existence of visceral disease in nervous affections. The infectious diseases are especially liable to affect the nervous system, and in their course, as well as in the course of other bodily disorders, there must be constant watchfulness of the cerebrospinal axis, particularly during the acute stages and during convalescence.

The diseases of the ductless glands deserve special attention. Their earliest manifestations ought to be recognized and adequate measures taken to prevent the later development of system affections of the nervous and muscular systems. As for the avoidance of physical and mental overexertion, its significance is so evident that it need only be mentioned.

Sufficient recreation, sleep, and variety in work are of obvious value.

GENERAL THERAPEUTIC AGENTS

Any thorough workman about to undertake an important task must make a study of his tools, noting carefully their possibilities and their limitations. So, also, before we take up the study of the treatment of the individual muscular diseases, the means of therapy, exclusive of medication, ought first to be considered, in so far as they bear immediately on our subject.

The principal forms of therapy are electrotherapy, hydrotherapy, and mechanotherapy.

Electrotherapy.—Great skepticism exists as to the therapeutic value of electricity in all diseases. Nevertheless, an unbiased critic, putting aside all results due to suggestion, can point with truth to the markedly beneficial effects of electricity in the treatment of atrophies of muscular, of neuritic, and of arthrogenous origin. Equally beneficial results can be demonstrated in conditions where suggestion evidently plays no part. There can be no doubt that electricity is a very useful therapeutic agent, in proper cases, when correctly applied.

Three kinds of electricity are commonly applied in therapy:

1. The constant or galvanic current.
2. The induced or faradic current.
3. The high tension or Franklin current.

We must remember that uninterrupted application of the cathode acts as an excitant, useful where centres or nerves show diminished irritability. The anodal is a sedative, non-stimulating current, and is useful in irritative conditions, motor or sensory; rapid alternations are contraindicated where these effects are desired.

Galvanic Current.—The doses of the excitant cathodal current ranges from 2 to 5 ma., the dose of the antineuralgic anodal application 0.5 to 2 ma., size of electrodes 4 to 5 cm. in diameter: application to any given site about two minutes: total duration over many places ten minutes: frequency of application once daily. Only in special cases is a more frequent, or longer continued application desirable. Such prolonged applications are especially valuable for neuralgias. Galvanism should be given a fair trial, and if within a few weeks no distinct improvement is apparent it should be discontinued.

Faradic Currents.—Weak faradism increases and strong diminishes irritability, but, owing to the painful character of this current, only weaker stimulating and tonic currents should be employed. An electrode of 4 or 5 cm. diameter, rounded, connected with the negative pole, should be applied locally, each single application lasting two minutes: total duration of entire séance ten minutes.

The faradic current is of distinct importance in paralyses after disuse, muscular wastings following dislocations, fractures, tenotomies, and arthritides. Incidentally it is efficacious in sensory disorders, and for its reflex effects.

Leduc's intermitting currents of low tension, 40 interruptions of the galvanic current per minute, have a powerful effect on muscles and nerves, without sensory effects. It acts beneficially on degenerated muscles, and has a greater penetration than the ordinary faradic current.

Galvano-faradization.—De Watteville praised this method of combined application; the favorable action of the galvanic current acting as a tonic to fatigued muscles, compensates the exhausting effects of faradism. We must bear carefully in mind that, where the reaction of degeneration is present, strong currents are to be avoided.

The electrolytic, cataphoric, and vasomotor effects of electricity may be summed up briefly. They aid absorption and favorably influence nutrition. The galvanic current is employed; the anode in acute and exudative processes, the cathode in chronic torpid processes, with diminished circulation, degeneration, and sclerosis. The anode in painful conditions, the cathode in painless states, thus permitting the employment of much stronger currents.

For Franklinization, sinusoidal, and d'Arsonval currents the reader is referred to special text-books, as their employment is very limited in the muscular atrophies. It is true that Franklinization has been employed in muscular atrophies, palsies, and rheumatic affections, but I have never seen any good result from its use.

From this brief general consideration of the subject of electricity in its application to the muscular atrophies it is obvious that the galvanic and faradic currents appropriately applied are of decided benefit.

Hydrotherapy.—It is my object to give only a general account of the *rationale* of hydrotherapy in its application to muscular atrophies. Water is used as a convenient medium for the application of various

physical forces, such as heat, cold, pressure, etc., to the body. The intensity of the reaction depends upon the intensity of the force applied, and the site and extent of the application. The intensity of the force depends upon the amount and the duration of the application. The effects depend upon the rapidity of temperature alterations, and upon local and general individual differences. We must bear in mind that weak and medium irritations are ultimately stimulating; extreme irritations ultimately depressing, and that the various sensibilities are differently affected by heat and cold. Vinaj, after accurate experiments, determined that cold increases muscular capacity and warmth diminishes it. All hydrotherapeutic effects act as nerve irritants, the intensity of the stimulus being increased by differences of temperature between the water and the surface temperature at the site of application, by the extent of surface, by the duration of application, and by the pressure employed. The more sudden the application the greater the stimulus; and similarly the more hyperesthetic the skin the greater the reaction. By increasing or diminishing the irritation we can increase or decrease the reaction. Too intensive irritation inhibits or may even completely paralyze reaction, but weak and moderate applications stimulate. These effects are produced by thermic and pressure irritations of the sensory end organs and peripheral bloodvessels, which modify the local circulation.

Mechanotherapy.—Of the various modes of administration of mechanotherapy the first is massage. The physician, by means of various manual movements, exerts mechanical action on muscles at rest. I say advisedly the physician, because in the conditions to be taken up later the masseur must be fully cognizant, at all times, of the condition of the muscles under his fingers, and must be fully acquainted with the underlying pathological process. Indiscriminate massage, ignorantly applied does more harm than good, and frequently brings about greater injury to the affected muscles than already exists. The time has passed when this valuable therapeutic agent should be confined to the hands of those celebrated only for their great strength and not for their knowledge and skill. It is no more undignified to apply proper massage than to apply electricity or any other therapeutic agent. Along with a practical knowledge of hydrotherapy, the ability to apply massage should be a part of the equipment of every physician today.

Massage.—Massage is of special service in atrophic muscular states and deformities resulting therefrom. It increases the nutrition of muscles made inactive by immobilizing apparatus and exerts a beneficial action on their circulation and the local metabolism. It restores function to muscles where gymnastics are impracticable or contra-indicated, and diminishes cyanosis, edema, and local heat. Moreover, it has a sedative action (when properly applied) on irritable tissues, acting as a preventive of spastic contractures due to local pain, and aids the resorption of the residues of old inflammatory processes in joint effusions, especially after operative interference. It diminishes or prevents deformity following scar formation.

Gymnastics.—Next in importance to massage comes gymnastics—the methodical exercising of motor organs with especial reference to local effects by simple active, simple passive, complex concentric, or complex eccentric movements. The active movements or exercises are (individually or in classes) performed by the patient himself, without outside assistance, *i. e.*, by his own muscular innervation, as in turning, balancing, etc., such movements being useful for their effect on general nutrition. Variety and quality are of importance. Their purpose is to cause the muscles to respond quickly to the will. Their moral value is evident. Every movement means functioning, local hyperemia, and increased local and general metabolism, with increased nutrition, increased strength, and finally an increase of the contractile elements as consequent results. Overdoing must be avoided; there must be individualization; fatigue should be prevented by moderation, by intermissions and by variety in exercise.

Passive movements are performed without any volition on the part of the patient—exogenous exercises so to speak—and may be performed by the physician himself, without the aid of weights or machines. These movements are indicated in contractures, and joint deformities, to prevent or break down moderate adhesions, and to keep the tendons and muscles freely movable in their sheaths. They mainly affect the elasticity of the muscles, and with the changing tension salutary effects on the circulation and nutrition result.

Of great value are the complex movements—the resistance, or so-called Swedish movements. The addition of volitional effort enhances the value of the passive movements, but the resistance must be gradually and carefully applied by an experienced physician. They may be concentric, *i. e.*, active flexion against resistance; or eccentric, *i. e.*, passive flexion against resistance. The movements must be smooth, uninterrupted, gradual, and fully adapted to the patient's strength.

To dispense with the need for an operator various apparatus, known as the Zander apparatus, have been devised. By their use, exact dosage of the resistance and precise regulation of the amount of force to be expended by the patient is possible. These, used under the watchful and skilful care of the physician, are the last words in mechanotherapy.

There are two main varieties of apparatus: (1) Those moved by the voluntary effort of the patient, (2) those operated by a motor.

Three varieties of movements may be accomplished by their use:

1. Movements for the exercising and developing of muscles.
2. Passive movements, without the assistance of the patient, to stretch capsules, tendons and muscles, and to soften them.
3. Movements for mechanical effects, such as shaking, kneading, chopping, rubbing, pulling of muscles, *i. e.*, massage.

There are also in existence various kinds of apparatus, by the aid of which the patient can exercise himself, and bring about similar results. Experience, however, teaches that, for various reasons, too much as a rule cannot be left to the patient.

In many cases the exercises, which I have described, are insufficient to bring about correction of contraction or deformity. In such cases various operative procedures, such as *redressement forcé* or other forcible manipulations, under anesthetics are necessary. At times mechanical contrivances are imperative.

For the various operative procedures, such as tenotomies, muscle transplantation, nerve transplantation, and arthrodesis, the reader is referred to works on surgery.

PARALYTIC CONTRACTURES AND ANKYLOSES

The commonest complications of muscular atrophy are the paralytic contractures and the ankyloses, and a study of their pathogenesis ought to precede the study proper of the atrophies.

To understand paralytic contractions, the study of the action of antagonistic muscles is necessary. This is best studied after a tendon has been divided. When the tendon has been completely separated from its bony attachment, a permanent contraction of the muscle ensues; it retracts like an overextended elastic band. Owing to the poverty of the elastic substance and its lack of firmness, the muscle can still be stretched for a given time. Then shrinkage takes place, and ultimately the muscle cannot be stretched any longer. In the earlier stages, no histological changes are to be demonstrated, but finally a *myositis fibrosa* results.

But with the antagonist a different state of affairs ensues. The antagonist contracts the instant the muscle tendon is cut; its own elasticity keeps it contracted. It does not shrink to the extent of the opponent, because it is frequently but involuntarily put on stretch. If a finger tendon be cut, contraction of the other fingers will cause concomitant contraction of this muscle automatically, thus preventing atrophy from disuse. But, as we shall see later, if it be immobilized, atrophy will ensue, unless passive movements are instituted.

A completely paralyzed muscle acts like one with a severed tendon. The ordinary weight of the part is insufficient to overcome the contracture.

Muscle elasticity is often confounded with muscle tone. The latter is a constant, weak, involuntary contraction of the muscle dependent upon the integrity of the nervous system.

Various theories have been advanced to explain the occurrence of contracture in paralyzes, but most probably it is, as Seeligmüller believed it to be, a combination of the action of the antagonist and other mechanical actions. He believed that in all cases of paralysis of joint-moving muscles voluntary impulses only reach the non-paralyzed muscles, moving along intact neural elements. As a result the non-paralyzed muscles will alone act under volition, the limb will assume one position, and remain in that attitude by reason of the inability of the paralyzed muscle voluntarily to contract, and a shrink-

age in this muscle ensues. Every new volitional impulse will take the same course; shrinkage becomes extreme, aided by the weight of the parts; as paralyzed plantar flexors, in *pes calcaneus*. Where these contractions do not develop it is due to the occurrence of passive movements in the paralyzed muscles. What appears to be a central predisposition to contractures can be recognized by vasomotor phenomena like edema in the paralyzed part.

Briefly, contractures are due to voluntary contractions of the antagonist, and not to the tonus of the non-paralyzed muscles. The terminal attitude depends on other factors, those of the weight of the limb and the burden put upon it, in other words, on mechanical influences.

The instant a muscle is paralyzed, the spasm of the antagonist begins. Which factor predominates must be considered here.

When all the muscles of a joint are totally paralyzed, a flail-joint results where no mechanical movements are employed. If the latter are instituted then a contracture ensues, the type depending upon the usual position given the part, in bed or by splints.

If the paralysis be incomplete, but fairly equalized, so that some muscle function still persists, then the influence of the weight of the limb in standing, walking, crawling, pushing, etc., plus the physiological preponderance of the flexors over the extensors will determine the form of contracture.

When certain muscle groups are involved, then the contracture is ushered in by the contraction and elasticity of the antagonists. The more closely approximating points of insertion are never separated to their former distance, and a nutritional shrinkage ensues that can even overcome the effect of the dead weight of the extremities, and also the burden put upon it by the rest of the body. The weight and incumbrance acting together may increase the contracture, but if applied antagonistically to one another may exert no influence on the production of contracture. Generally the external forces act more powerfully than the functioning muscles in effecting the production of contracture.

Trophoneurotic factors, especially in growing children, act deleteriously on the muscles previously shortened.

Ankyloses.—Ankyloses are frequently sequelæ to contractures. They are states in which two or more joint surfaces, owing to local or intra-articular changes, become fixed and immobile, and unite. The new interarticular tissue is usually connective tissue, especially in the early stages, and if soft and pliant forms what is called an incomplete ankylosis. In the course of time, changes in the cartilages result; the latter disappears and bloodvessels form in the new tissue—the so-called ankylosis fibrosa cartilagina. Where cartilage remains plentiful, then a cartilaginous ankylosis develops. After a still longer time, a union of the joint surfaces occurs and an ankylosis fibrosa interossea results. Last of all an ankylosis ossea, in which the new bony tissue originates from a metaplasia of cartilage or from outgrowths from the bones themselves. Mixed types also exist.

Treatment.—These ankyloses do not improve without treatment, but rather tend steadily to increase and finally become stable. If secondary inflammation occurs, the ankylosis may become bony.

In their early stages, if we except those of developmental origin, they are curable; at least, a restoration closely approximating the normal may be produced. A completely paralyzed muscle cannot be fully restored, but a considerable restoration of power may follow suitable treatment.

If at the onset adequate and proper means are applied, the normal static relations may be restored, and the bony development will then follow along normal lines. Yet it is just the early stages that are overlooked or improperly treated. Mechanics in orthopedics are useful when working under skilled physicians' orders, but their independent efforts are fraught with danger. A contractor is never called upon to take the place of an architect; so in this more important branch a brace-maker is neither a diagnostician nor a proper prescriber of orthopedic apparatus.

The treatment should be (1) preventive, (2) corrective, and (3) directed toward prevention of recurrence.

In all joint conditions, where deformities are apt to occur, the patient must be placed under such conditions, at rest or in bed, that ankylosis may be avoided or, when it is inevitable, that the resulting position of the part shall be the most serviceable. The same problem confronts us when we deal with contractures. Apparatus must be employed where necessary; parts must be placed in the most favorable positions. They may have to be placed in extreme opposite directions when we have to deal with scars that have a tendency to contract, and by their shrinkage to cause deformity.

Where hereditary tendencies exist, children must be carefully watched, both at home and at school, for the earliest evidences of hereditary disease. Proper exercises, gymnastics, etc., are very useful. The remarks on the hygiene of childhood hold here with special emphasis.

For information as to the various corrective apparatus, the reader is referred to text-books on orthopedics.

Prognosis.—The prognosis of ankylosis depends upon its duration, the degree of deformity, and upon proper skill in treatment. We must always have in mind a clear conception of the underlying pathological changes. Marked deformities do not of necessity mean a poor prognosis; the essential point is whether the ankylosis is congenital or not.

MUSCULAR ATROPHIES

Muscular Atrophy in General.—Muscular atrophy is a clinical entity only, and to understand it a thorough study is necessary.

The first cause is trophic. Aside from its blood supply, a muscle depends for its integrity on the lower motor neurone; that is to say,

the ganglion cell in the anterior horn of the spinal cord (the true trophic centre), the axis-cylinder, and the end spindles. The mechanism of the lowest synapse between the muscle spindles and the muscular fibers is not as yet clearly known. Function is, we know, carried along the same paths.

Whether or not other trophic centres exist in the Rolandic area, basal ganglia, etc., is still an open subject, as the study of cerebral and arthrogenous atrophy will show. The generally accepted view is that the true trophic centre lies in the anterior horn cells, but that this control is regulated by stimuli from the periphery and from the central cortex. It is not necessary to formulate a cerebral centre as Quincke has done, but only to account for muscular atrophy in cerebral disease as due to the abeyance of the stimuli regulating the trophic centres.

Not alone the absence of volition, but the absence of motor impulses also serve to diminish these controlling stimuli; these are best seen in hemiplegias.

The ganglion cells remain intact as long as reflex activity exists. When, however, the disease spreads from the degenerated pyramidal tracts to the anterior horns, then severe trophic changes result.

There are also peripheral or reflex atrophies, due to irritating joint diseases. The excess of sensory impulses brings about muscular atrophy, which may be avoided by cutting the posterior roots. While this explanation generally holds in individual cases, a combination of causes may occur, *i. e.*, aside from joint conditions, such as a preëxisting injury to the anterior horn cells, which has left in its wake a diminished resistance. Steiner speaks of a lessened autonomy of the spinal centres, which can persist in an infantile state. Such a ganglion cell will naturally suffer more from the abolition of volitional and other impulses. This probably explains the severe atrophies seen in juvenile hemiplegias. The same explanation may also pertain to the arthrogenous atrophies. The selective action of various toxins, such as lead, and alcohol on the anterior horns is well known. The muscular atrophy seen in hysteria may be explained by a diminution of tone in the anterior horn cells.

The other group of atrophies is due to primary or secondary disease of the anterior horns themselves, and the essential spinal types of muscular atrophy as seen in poliomyelitis, amyotrophic lateral sclerosis or syringomyelia, and the neural atrophies seen in neurotic muscular atrophy, and the various polyneuritides.

It must be borne distinctly in mind that the separation of a muscle from its nutrient cell does not, as in the case of a nerve, bring about a degenerative atrophy. There occurs a disappearance of muscle fibers, a simple atrophy. A muscle is not an integral part of the lower motor neurone, but is a distinct entity, and the loss of function is similar to the loss of neural function in supranuclear disease. Other factors are necessary to cause a degenerative muscular atrophy, *i. e.*, changes in the circulation of the blood and lymph, inflammation and the specific action of degenerated anterior horn cells on the muscles themselves,

the nerves connecting them remaining uninvolved. The *rationale* of this action is not definitely known.

Reflex Atrophy.—Reflex atrophy, another variety, may be due to two mechanisms: (1) a vasomotor influence bringing about nutritional changes, or (2) through the direct action on the trophic centres in the spinal cord. The second is the generally accepted explanation. Charcot has distinguished two forms: an abarticular and an articular. He has also differentiated the stages of the latter, of stupor and of inertia, which is very transient, and may disappear very rapidly under electrical treatment.

In this stage no histological changes in the cord are perceptible; but after long duration irreparable deteriorations cause atrophy, and a diminution of ganglion cells occurs, while in extreme cases total disappearance may result.

These changes can be brought about experimentally by injection of irritants, such as mustard, turpentine, silver nitrate into joints, from which atrophy results, whether the parts be moved or not. If, however, the posterior roots be cut, atrophy does not take place. These experiments show fairly conclusively that the muscular atrophy is due to action on the cord itself. Brown-Séquard believed that the irritation was caused by vasomotor inhibition, which ceased when the roots were cut. The occurrence of atrophy in long-standing cases would appear to negative this, however.

Reflex atrophy has the following characteristics: a short time after the accident, flaccidity and a myasthenia appear, then exaggeration of the tendon and superficial reflexes, increased myotatic irritability, increased faradic excitability, and sensory phenomena appear. Rapid diminution of volume occurs, which when properly treated as rapidly ceases.

In addition, the atrophy bears no direct relation to the amount of muscular activity, to the loss of power, or to the nature and extent of the injury. It has a selective action on the entire length of the extensor muscles of a joint; there is only a quantitative change in electrical excitability, and histologically only a simple atrophy results.

We shall consider below in greater detail the clinical picture and the therapy of reflex atrophy.

Inactivity or Disuse Atrophy.—Normally the muscular volume is proportionate to bony development, and to the ordinary work performed by the muscles, though naturally a wide margin of physiological variation exists. Formerly too great importance was attached to the role of inactivity in the production of atrophy; yet its influence in old dislocations, in ankyloses, after tenotomies, in patellar fractures, and in amputation stumps is unmistakable.

Klippel showed that in ankyloses of the knee the quadriceps was unequally and irregularly implicated. The vasti with great limitation of motion, showed the maximum atrophy, while the rectus femoris which still passed over a functioning joint was affected to a much less extent. Hanau showed similar selection in ankyloses at the ankle.

For the proper nutrition of a muscle there must be some activity, if ever so little.

In amputation stumps, those muscles atrophy most which do not pass over a functioning joint—in the leg the soleus most and the gastrocnemius least, and in the thigh, the vasti more than the recti.

In atrophy from immobilization under cast or brace the complicating influences are pressure by the cast, or the initial injuries to the muscles at the time of the fracture. Bone and tendon diseases have a concomitant deleterious action. Inactivity atrophy is an undesirable complication. The proof of the causative relation of inactivity is the absence of atrophy under electrical or physical therapy. Even in primary muscular atrophies inactivity plays an important part, a fact which must be borne in mind when we come to treat of these atrophies. Scores of instances of inactivity atrophy can be mentioned. The waste of those muscles most employed in health, during prolonged confinement to bed, must have impressed every clinical observer, yet, peculiar to relate, careful microscopic examination fails to substantiate the existence of this specific variety of muscular wasting.

As the antithesis to the above is the muscular hypertrophy due to activity. Nothnagel, with frogs, and Morpurgo, with a dog in a treadmill, were the first definitely to show this increased volume; this phenomenon has since been studied by many other observers. The hypertrophy is due to increased functional stimulation, resulting in dilatation of the smaller bloodvessels of the muscles, and in increased capillary and lymph circulation following the increased muscular action; but although these factors are of great importance they are, nevertheless, not fundamental, Nothnagel believed that a specific cellular activity was responsible.

Increased waste and repair, to the physiological boundaries, takes place in the sarcoplasm, leading in a short time to an increase in the diameter of the muscle fibers, and an increase of fibrils; there thus results a true hyperplasia. If the physiological boundaries are exceeded, overuse of the muscle substance occurs, and damage results to the muscle fibers. The older and less actively functioning ones first show defects and lose their function.

Under favorable nutritional conditions an overproduction of muscle cells takes place and muscle fibers, depending on the functional activity, either develop or die. With long-continued yet physiological activity, and with good nutrition there results another variety of hypertrophy—the numerical hypertrophy or hyperplasia. With poor nutrition, with excessive or inefficient functional stimuli, the new elements as the oldest fibers perish, and a numerical atrophy ensues.

The amount of muscular damage in inactivity atrophy is proportionate to the disturbance of function. In transient and incomplete inactivity a simple atrophy without loss of fibers results. Such muscles become normal on the restoration of activity.

In long-standing cases of incomplete atrophy, only single muscle

Fig. 1



Muscular Wasting in Chronic Heart Disease.
(Montefiore Home)

PLATE I

Fig. 2



Muscular Atrophy in General Osseous Tuberculosis.
(Montefiore Home)

fibers waste, and restitution becomes doubtful. Normal regeneration remains in abeyance in the inactive muscles.

Under complete inactivity, as seen in immobilization, degeneration follows, with loss of fibers, and more or less extensive atrophy. Here regeneration is impossible, and restoration of function causes hypertrophy and hyperplasia of the remaining fibers.

In youthful individuals, however, bioplastic changes occur which accelerate the disappearance of muscles. Here, in the absence of the natural stimuli to muscular growth, the newly formed cells, instead of developing into new muscle, degenerate until finally the entire muscle disappears and is replaced by fat. Complete disappearance never occurs, however, as isolated muscle fibers are found within the fat of aged, unused stumps.

Influence of General Nutrition on Muscular Atrophy.—Profound disturbances of nutrition act deleteriously on the contractile substance, and can directly or indirectly bring about simple or degenerative atrophy. The two main sources of nutritional disturbance are circulatory disorders and general inanition. In long-standing cases, the former brings about granular, fatty, fibrillar degeneration. In transient ischemia the changes are only partial. These products of degeneration are rapidly absorbed, and a loss of muscular volume results, the extent of which is dependent upon the degree of degeneration existing. With the return of circulation these signs disappear and there remains, according to the intensity of involvement, either simple atrophy, or numerical atrophy, or, in extreme cases, even muscle defects.

In inanition simple atrophy is found at the onset; but later a degenerative atrophy results. The muscle disappears simultaneously with the fatty subcutaneous tissue.

Experiments made on starving cats show a loss, within thirteen days, of 30 per cent. of the muscle volume, and in slow starvation a still greater atrophy is possible. Gaglio experimenting with the gastrocnemii muscles of frogs found, at the end of a year, a loss of 85 per cent. in volume, the atrophy being not so great in the heart. It must be borne in mind that muscles are not alone organs of motility, but are also storehouses for proteids, carbohydrates, etc. Inanition shows an affinity for the sarcoplasm, and then for the fibrils, the nuclei remaining unaffected. The narrow protoplasmic fibers are primarily involved, the wider later and to a less extent.

The pathological anatomy shows three stages; a stage of narrowing of the fibers, a stage of clouding of the muscle substance, and a stage of small-celled, and, later, of large-celled infiltration of the contractile substance. These greatly resemble fatty degeneration, yet staining shows no true fat.

In human beings, inanition is always combined with a febrile toxemia, which in itself exerts a selective action on muscles. Experiments on fasters show a marked muscular wasting.

Influence of Stretching and Constant Pressure. Many experimenters have shown the existence of muscular atrophy after overstretching.

Kramer placing an inflated bag under muscles produced a wasting of 25 per cent. in eight days. This accounts for the atrophy of the deep spinal muscles on the convex side, seen in cases of extreme scoliosis; also the atrophies in club feet, etc. When a muscle is diseased, the deleterious effect of overstretching becomes more evident, as seen in the quadriceps wasting in arthritis of the knee-joint, where there is extreme flexion of the leg.

Constant pressure on a muscle exerts a similarly baneful influence. In this case there results a local ischemia, interfering with metabolism, and also with function. The application of electricity will prevent the atrophy.

Clinical Symptoms of Muscular Atrophy.—These are initially weakness and atony. The muscles feel soft and doughy and a diminution of volume becomes apparent, but pain and local tenderness are only found where complications exist, and when the atrophy is very rapid.

The atrophy may be local or diffuse, the extensors commonly being the more affected. Loss of function and muscular wasting run parallel.

In the early stages, electrical examination shows a normal or even an increased irritability. Later, depending upon the intensity of the disease process, the irritability diminishes steadily, and in severe cases may be abolished. Qualitative changes, partial or complete reflex degeneration, are found only where the lower motor neurone is also involved. An exception to this rule may occur in cases of cerebral atrophy. Pettina, in a case of Jacksonian epilepsy, found an increase of indirect galvanic and faradic irritability, and a diminution of direct muscular irritability. Myotatic irritability is increased and idiomuscular bundles are commonly observed.

We shall now consider under separate heads the varieties of muscular atrophy.

Constitutional Disease as a Cause of Muscular Atrophy.—The constitutional diseases which may be causative of muscular atrophy are:

1. Acute or subacute infectious diseases, such as typhoid, cholera, septicemia, pyemia; but exclusive of diseases combined with multiple neuritis, such as diphtheria. Some include here trichinosis. A symptomatic myositis is found in these cases.

2. Chronic febrile diseases such as malaria, tuberculosis, etc. In these regional atrophy, especially of the thoracic muscles, is common.

3. Chronic cachectic diseases producing inanition, such as senile involution; chronic digestive diseases, and malignancy.

4. Exogenous intoxications, such as lead, carbon dioxide, etc. Lead may lead to diffuse, widespread atrophy.

5. Auto-intoxications, pellagra, cachexia, rickets, and Basedow's disease.

Clinical Pathology and Course.—In the third group, we find a general and equally distributed atrophy with defective regeneration, typified by the wasting of the muscles of the back seen in the senile.

In the second group, of chronic febrile states, like tuberculosis, either

PLATE II

Fig. 1



Muscular Atrophy in Cancerous Cachexia. (Montefiore Home.)

Fig. 2



Muscular Wasting due to Pott's Disease. (Montefiore Home.)

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a local or widespread atrophy is found. The pathological anatomy is a simple atrophy plus degenerative atrophy plus regenerative changes. Myalgias are frequent in these cases.

In the various atrophies due to intoxications, as the localization and spread depend upon the specificity of the toxin, the atrophy may be general or local. They are very often accompanied by severe myalgias.

Arthrogenous Atrophies.—These atrophies are mainly reflex atrophies, and all that has been said above applies to them. Clinically speaking, two types may be differentiated, the traumatic form and the inflammatory form. The latter group may be acute, subacute, or chronic, and includes rheumatic gout, arthritis deformans, tubercular and luetic joints. The clinical picture differs chiefly in the intensity of the atrophy and not in the main clinical symptoms. It reaches a maximum intensity in ankyloses (*vide supra*).

Pathogenesis.—The pathogenesis of these atrophies is still *sub judice*, some authors believing them to be due to immobility others to deleterious pressure, others again to local hyperemia, and still others to the spread, by contiguity, of the inflammation, while others again think peripheral nerve changes are responsible, and report such present. Later observations have failed to corroborate this. In tubercular joints the local toxic anemia of the muscles was long held responsible for the wasting, but experimental injections of irritants did not produce the characteristic wasting, and the beneficial results of electricity and massage are not such as would arise for a muscle the seat of a focal irritation.

Then the French school found that the atrophy was of reflex origin, and due to changes in the anterior horn cells. The mechanism of this change was not clearly understood, some believing it to be due to vasomotor influences, and others to a direct injury to the ganglion cells. Investigators found anatomical relations between the nerves of the joint capsule and the extensors acting upon the joint. Thus was explained the fact that the extensors were mainly involved in joint affections.

Then the association of the maximum tension within the joint, and the action of the extensor group of muscles in increasing intra-articular tension was thought to stand in causal relation to the atrophy of these extensors.

Lorenz believed that the influences acting are many, and that they act in various ways.

1. There may exist a true reflex atrophy, as in the experimental and traumatic types; also occurring in some inflammatory conditions. Here, without limitation of movement, with minimum joint affection, a rapid onset of muscular atrophy takes place, which under improper treatment, may exist after cessation of the joint affection, or may progressively develop. Under proper treatment, in these cases, the atrophy can be made to disappear entirely.

2. In chronic purulent or fungoid arthritis the inflammation may be spread by contiguity, and may directly involve the neighboring muscles, causing degenerative changes in them.

3. In ankylotic states, or those in a stationary condition, marked with only partial ankylosis, an inactivity atrophy of slower development occurs.

Symptoms.—The clinical picture of arthrogenous atrophy may be summed up briefly. The initial symptom is a feeling of weakness, or an actual paresis of the affected limb. The muscles feel soft and flabby; the muscle tone is diminished, the normal contour is changed, and a partial contracture is produced.

This condition has been variously spoken of as muscle collapse, or psychic impotency of the muscle. Sensory changes are rarely present. Actual changes occur during the second week, or earlier, depending on the severity of the joint affection. This atrophy advances rapidly.

FIG. 11



Muscular atrophy in spondylitis deformans. Note extreme rigidity and the marked atrophy of the pelvic and thigh muscles. (Montefiore Home.)

Its time of maximum severity is variable, and may occur even after the joint condition has passed away, or, as in inflammatory cases, during the latter days of the disease. The extensors are first involved, and a flattening is soon visible. In arthritis of the knee-joint, the quadriceps, especially the vastus internus, is involved; in the shoulder, the deltoid and scapular muscles are chiefly affected. There are no evidences of pain nor local tenderness in the muscles; electrical changes, however, are found in the shape of diminished faradic irritability, at times diminution of irritability to galvanism. In individuals with neuropathic taint, however, the atrophy may spread beyond the usual confines. At this time contractures may be found, especially in tender joints where spasms *de défense* exist. These spasms may be

intermittent or continuous, and shortening of the muscles may occur. Permanent contractures occur only in chronic cases.

Course.—The course of the disease depends on the severity of the pathological process, and recovery may be astonishingly rapid where the disease is not of long standing. In the latter case there exists a tendency toward further progress, and the atrophy remains localized with the exception that the antagonists may also be involved.

In the chronic forms a progressive muscular atrophy may be simulated, as in cases of spondylitis deformans, and arthritis deformans, which I have personally observed. Here the clinical picture resembled very closely that of an advanced progressive muscular atrophy. Similar changes have been observed in chronic gout. Cornillon found atrophy of both deltoids, and of the extensors of the fingers, interossei, thenar, and hypothenar muscles with flexor position of the terminal phalanges of the hands. Curschmann, in a juvenile arthritis deformans, found atrophy of the hand muscles, also arm and shoulder atrophy, resembling an Erb palsy. Dejerine reports cases like a myopathy; Rummo, an Aran-Duchenne picture. Extensive atrophy occurs mainly in children. Moristin speaks of a dystrophy arthropathique; Achard and Levi found secondary atrophy of the upper motor neurones (Figs. 11 and 12).

Treatment.—The treatment must obviously be based upon the nature and cause of the underlying morbid process. Le Fort employed weak permanent currents; in severe cases continuously, day and night, in milder cases, only at night. He noted marked improvement from the second to the fourth week. Faradism plus galvanism, *i. e.*, galvanofaradization, is still more efficacious. The most important factor in therapy is the early establishment of muscular action, partly by means of electricity, and partly by massage and by resistance movements. In recent cases cure is fairly certain if the patient contributes voluntary movements. Writers disagree as to which of the several agents—electricity, massage, or active movements—is the most efficacious. When atrophy has developed, and the arthritis has ceased, energetic massage, and resistance movements are of great value. In extreme or old-standing cases no treatment avails.

Muscular Atrophy after Accident.—In taking up this subject at this time, we have to bear in mind the clinical resemblance between

FIG. 12



Same patient as Fig. 11.

this type of atrophy and arthrogenous atrophy. The diffuse type will be taken up again later in the study of progressive muscular atrophy.

In the diffuse type, as is common in other injuries, such as traumatic pneumonia, there is little or no evident injury to the parts. Charcot speaks of this form as the abarticular type of atrophy, and Lemcke, as a traumatic insufficiency. A patient has a trivial trauma to the knee, and we notice after a time a progressive wasting of the quadriceps femoris. Immediately after the injury, nothing is apparent, and the knee is freely movable, but after a few days weakness develops, the muscle atrophies, following the same clinical course as in the arthrogenous type; the muscle becomes soft and flabby, and then atrophy becomes evident. The loss of function is never complete. Electrically one finds a diminution of faradic irritability. Hydrops of the knee is often present. In Charcot's case there was a trauma, with overstretching of the quadriceps, by a fall on the knee. In Riedinger's case, a similar injury resulted in a flail-joint.

A reflex atrophy is here improbable, because the inflammatory changes are so inconsiderable as to be negligible, and there is very little limitation of joint motility, and the muscles show no inflammatory changes. Nevertheless a rapid atrophy follows, which Ballet and Bernard called an atrophy *en masse*.

Désnos and Barie found a general atrophy of the entire limb in one case. Picqué found extensive atrophy after a superficial injury. Rummo, in 1898, after a fracture of the clavicle and olecranon found an atrophy of the Aran-Duchenne type, which some authors believe to be not uncommon.

The clinical picture is a flattening of the muscles, diminished electrical irritability, atony indicating a loss of gross motor power, and diminution or loss of the volitional power.

In neuropathic individuals, suffering much pain, the loss of function can be partly accounted for.

Quillain found bilateral atrophy, and in Jolly's case there was a preëxisting poliomyelitis.

It must here again be noted that in growing children there is a greater liability to extensive muscular wasting. In the localized forms the trauma causes more or less destruction, or injury to muscle fibers, the amount of atrophy being proportionate to the amount of local injury.

In this group we must include the pressure atrophies, and the occupational pressure atrophies. These will be discussed in the chapter on the occupational diseases.

Treatment.—The treatment of the diffuse, and of the localized forms, is similar to that of the arthrogenous forms; but, in addition, strychnine may be given hypodermically in cases in which the wasting is widespread or of long standing. The details of treatment will be found under the heading of the muscular atrophy of spinal type.

I desire briefly to call attention to muscular atrophy following bone and tendon injury; (1) such as is seen in fractures where the

mechanism is complex, and many factors are responsible for the wasting; (2) after tenotomies, where atrophy follows a brief hypertrophy; and (3) muscular atrophy after amputations.

Here the treatment is along the lines already outlined for inactivity atrophies.

Muscular Atrophy following Overexertion.—In considering this group of muscular atrophies, we exclude the atrophies following occupational neuroses, and occupational paralyses due to pressure.

Gessler has described the clinical course of this group. It begins with paresthesia, and cramp-like and paretic seizures. It is usually confined to certain groups of muscles, has no tendency to spread, no objective sensory disturbances, and the nerves are found to be normal. These cases are distinguished by their rapid cure under proper treatment.

In overexertion of muscles one would expect an hypertrophy. This is what does occur at first; yet the hypertrophied muscles are more easily fatigued or exhausted than normal muscles, and diminished regeneration takes place.

Treatment.—The treatment is cessation of the overexertion, moderate massage and passive movements, galvanic cathodal application of mild currents, or mild galvanofaradic currents.

Cerebral Muscular Atrophy.—This form of atrophy is generally regarded as uncommon. Yet anyone who has seen many cerebral diseases, especially hemiplegias, knows that it is not an infrequent, but a fairly constant symptom. Charcot, among many others, noted its repeated occurrence especially in old cases, and thought it due to inactivity. Some years ago I showed a case of this extensive early atrophy. Early cases, where no secondary spinal changes exist, are of the greatest interest.

This type of atrophy follows lesions in various parts of the cerebrum. It is improbable that there is a definite cerebral trophic centre. Some observers, however, attribute this trophic influence to the thalamus, others to the Rolandic area, others again to a general cerebral influence.

Von Monakow has laid stress upon the circulatory disturbances due to disease of the lenticulo-optic or the Sylvian artery. A cortical origin would explain the excessive atrophy seen in focal Jacksonian epilepsies of the motor area.

The extensive atrophy found in infantile cerebral palsy affecting muscle, bone, and subcutaneous tissue cannot be classified here, as in these cases a lack of development is mainly responsible. Another group not to be included are the atrophies seen in general paresis, as here spinal and neuritic changes also occur.

Pathology.—The pathogenesis is obscure and various explanations have been proposed. By some it is said to be due to an atrophy of the contralateral ganglion cells of the anterior horns arising from pyramidal tract degeneration. While this is undoubtedly true of the old cases, no such changes can be expected, nor found in the recent ones, as the degeneration of the pyramidal tracts at this time is inconsiderable.

Others believe it to be due to a dynamic change in the anterior horn cells which only later show microscopic alterations; but this has been disproved.

It cannot be an inactivity atrophy, because it appears too early in some cases; or appears after resumption of activity in other cases, and progresses in spite of activity.

The role it plays in older cases is marked by other complications such as overlooked arthropathies which develop insidiously without distinct subjective symptoms.

Some have assumed a cerebral trophic centre, others an involvement of the sensory pathways, and still others believe the origin to be vasomotor, and due to the inhibitive influence of the spinal vasomotor centres. In support of this contention they point to the vasomotor symptoms found in the paralyzed extremities accompanying cases of early atrophy. Others believe it to be due to a defective spinal autonomy; and so on, endlessly.

At present, it may be said that the late forms are of spinal origin, that the early forms are due to a diminution of the muscle tone, but that the majority of cases cannot be explained.

Symptoms.—Clinically this type resembles the arthrogenous. It may appear as early as the third day, and at any time up to eight weeks in the early forms. In the late forms the early contracture gives way to flaccidity.

It begins proximally, as a rule, and spreads to the periphery. The upper extremity, which is more under cerebral control, suffers earlier and more severely; most markedly about the shoulder-joint, especially the supra- and infrascapular muscles and the deltoids, the capsule of the joint becoming loosened. The pectorales are less involved. The upper arm may atrophy 2 to 4 cm., the forearm $1\frac{1}{2}$ to 2 cm. Similar figures are applicable to the lower extremities. In rare cases the lower extremities alone are involved. V. Bechterew found in a pons lesion an early atrophy of the leg only. Some have seen atrophy *en masse*. The atrophy affects muscles functionally related. Pain is rarely present.

Early atrophy is found with loss of muscular sensibility and astereognosis, yet no decided sensory changes need be present. Aside from a diminution of electrical irritability, the reactions are normal; fibrillation is rare; myotatic irritability is increased, and the tendon reflexes and deep reflexes are exaggerated. The progress of the atrophy is limited.

Treatment.—The therapy is the same as in the case of arthrogenous wasting.

Myositis Ossificans.—There are two types of this affection—a localized and a general multiple and progressive variety, the two forms being distinctly different.

Localized Form.—We shall first take up the localized form. In this type we exclude secondary ossification, with a starting point in the bone or periosteum, such as one finds commonly in the neighborhood of spontaneous fractures, especially of tabetic origin, *i. e.*, osteitis proliferans progressiva.

The localized form is mainly traumatic, due either to spontaneous muscular rupture, or to direct external violence, or it may result from repeated trauma of lesser intensity—the so-called exercise nodules. It is characterized by local inflammatory changes resulting in muscular nodes, which later ossify. The persistent or repeated irritations can easily account for the bony development, but in the acute form we must look to the irritation due to the presence of hematoma; the muscular activity constantly aggravating the pressure exerted by the hematoma. To explain its infrequency, we must assume that the individual is predisposed to such bony metaplasia.

The localized form is often seen in soldiers, hostlers, football players, etc.

The origin of the bone is a metaplasia of the interfibrillary connective tissue. The individual suffers a trauma, a hematoma develops, and there is local tenderness and pain. These subside, leaving a feeling of tenseness in the muscle and in three to four weeks later a bony nodule appears. The process is limited, partial absorption is possible, and a stationary condition results.

The treatment is surgical, and if a bony connection exists, a careful dissection from the bone is necessary.

Progressive Form.—Myositis ossificans multiplex progressiva is a multiple inflammatory process, progressive in character, affecting the muscular system, and occurring, in the great majority of cases, in youthful individuals. Its origin may date from the first month of life, or from any time up to fifteen years of age.

Etiology.—The etiology is obscure. The disease is congenital, non-hereditary, and is seen in individuals with a predisposition to calcification. The proof of its congenital origin lies in its very early development, its symmetry, its similarity in various cases, and its frequent and almost pathognomonic association with microdactyly. The bony deposits occur in the intra- and intermuscular connective tissue, and in the fascia.

Symptoms.—The clinical picture is typical. A local inflammatory change, usually due to trauma, marks the first stage. A firm, painful mass appears in the muscles. Occasionally slight fever and local edema and redness exist about this mass. In brief, the picture is that of a local myositis of varying intensity. These accompanying symptoms soon disappear, but the mass persists, resembling in adults muscular rheumatism. In this condition it may remain for a reasonable length of time, or else there may be a more rapid and diffused spread in the early stages. These masses are scattered, they spread, and later coalesce, until a considerable portion of the muscle becomes involved. Their consistency varies from that of a doughy to a bony hardness, but not all become bony; their size and shape vary very greatly. The various masses fuse together, and unite with the bones, and a marked impairment of mobility results.

The muscles most frequently affected are those of the neck and back, and in all cases the deep spinal muscles are involved sooner or later.

Fasciæ and bands and ligaments are included in the bony proliferation, the scapulæ become agglutinated, and the head becomes fixed. The upper extremities are usually involved before the lower, the masseters, and temporals may be affected, interfering with mastication, and the patient may ultimately become as stiff as a block of marble. The failure to implicate the muscles of the face, eye, tongue, pharynx, larynx, diaphragm, heart, perineum, genitalia, and sphincters is characteristic. In the early stages there is a diminished faradic response. Reaction of degeneration is rarely present. The urine shows diminished excretion of phosphates and lime salts.

The disease has a very characteristic course, requiring years for its full development, during which time remissions and exacerbations occur.

Treatment.—Efficacious treatment, as is evident from the description of the disease, is naturally very limited.

Baths and iodides have some influence in the early stages. When ossification is present they are of no avail.

Prophylaxis is very important. There must be a careful avoidance of injury to the unaffected muscles, which in children is naturally a very difficult matter.

Surgical treatment is followed by similar changes in the scars, and, except for jaw fixation, such treatment ought to be avoided. In some cases, total extirpation of the temporal muscles has been practised. Pollard and von Eiselsberg removed bony plaques with fair results.

Dermatomyositis.—**Etiology.**—This is an infectious disease due to toxemia by microorganisms, possibly gregarinidæ. It is a very severe malady with a mortality of about 50 per cent.

Symptoms.—It begins with prodromata—general malaise, headache, increasing general pains, and stiffness of the muscles of the back. Usually the onset is slow. A fairly high fever soon develops, and with it the characteristic edema of the eyelids and face. In time the pains become more localized, and the extremities show edema and the characteristic dermatitis—erythematous, erysipelalous, or urticarial rash spreading by continuity, steadily or in bounds, until a very large surface of the body becomes affected. The face, legs, and trunk look misshapen, and the edema is firm, and does not pit on pressure. The body of the muscle is involved, but the disease avoids the joints, especially those of the hands and feet.

The muscular pains are very severe, and are increased by the slightest movement, compelling the patient to be helpless in bed. Fever is remittent. The spleen is markedly enlarged, and perspiration is profuse. The superficial edema masks the muscles, rendering palpation futile. Nevertheless, there exists a decided swelling of the muscles. Every spread of the disease is accompanied by a rise of temperature. The muscles of the rump and the respiratory muscles are regularly involved; the intercostals, diaphragm, and even the pharyngeal and laryngeal muscles are affected. As a result the patient's state soon becomes critical, and death by suffocation or insufflation pneumonia ensues; or there may be marked remissions followed by exacerbations,

and the disease may last for months. Considerable atrophy takes place in these last cases. There are considerable variations in the course of the disease, and in the severity of the individual symptoms.

The constant signs are the characteristic edema, the localized swellings, the rash, and the limitation of movement. The patient, when the disease is fairly developed, looks typhoidal. In severe cases meningeal symptoms may arise. Pain, local tenderness, and paresthesia exist, but no nerve tenderness, and no objective sensory disturbance.

Diagnosis.—A case once observed is never forgotten, nor can the disease be even mistaken. Notwithstanding this, the resemblance between it and infectious myositis, metastatic abscesses, and septico-pyemia must be borne in mind. Trichinosis also bears a resemblance, but here the intestinal symptoms, and the finding of the trichinæ in the muscles serve to differentiate. The course of the dermatomyositis may be acute, subacute, or chronic, lasting respectively one to eight weeks, two and a half to six months, and one and a half to two and a half years.

Treatment.—The therapy is naturally limited. First and foremost one must bear in mind that this is an infectious disease, a toxemia, and treatment by elimination is of first and early importance. Thereafter the case must be handled like a typhoid, as to feeding, general hygiene, etc. Hydriatic measures, such as Pressnitz compresses are very useful locally. The respiratory embarrassment must be foreseen, and measures instituted to guard against such a complication. Among the drugs employed, sodium salicylate, antipyrine, and, especially in long cases, sodium iodide and thalline have been praised; but these drugs are merely agents which will fail us in severe cases.

Primary Myopathy.—A progressive, primary, usually hereditary disease of the muscular system, in which the muscles gradually weaken in a distinctive manner, and in which atrophy and hypertrophy and pseudohypertrophy coexist with fatty infiltration of the muscles.

The various types that have been described are clinical entities only, depending on different localization, and upon the predominance of atrophy or hypertrophy. The presence of both atrophy and hypertrophy, the course of the disease, the absence of other symptoms, the electrical reactions, the existence of transitional cases, and the presence of several types in the same family go to prove the essential identity of all. The other symptoms common to all are the rarity of fibrillary twitching and of reaction of degeneration. The electrical reactions are at first normal, but diminish steadily, until they are finally lost. Contractions are sluggish, and the amplitude of the galvanic curve is diminished. The tendon reflexes and the myotatic irritability slowly decrease and later disappear. The Achilles jerk, however, persists for a very long time. Sensation is intact, and the sphincters are very rarely affected. The sensorium is clear and dysphagia is extremely rare.

Contractures, due chiefly to shortening of the muscles and tendons, are very apt to develop. These contractures are of varying degree, and may become extreme, permitting no movement, even under

narcosis. They are not similar to those described before, because both synergist and antagonist are degenerated and paralyzed, and yet contracture may be extreme, nor are they due to difference in time of paralysis, because extreme contracture has been found with fairly functioning muscles, and, moreover, flail-joints are not uncommon.

Bone dystrophy also occurs, *i. e.*, an abnormal thinness, of the long bones especially. The skull and vertebræ are less frequently involved. Bulging foreheads, broad flat noses, bad teeth, clubbed fingers, short hands and feet, knock-knees, flat-feet, funnel-shaped thorax, pes equinus and equinovarus, claw-feet, marked lordosis—any of these may be present, and the deformity may be extreme.

The disease occurs in early or late childhood, at puberty, less frequently between puberty and forty, but later is very rare. It is essentially familial, two or more members of a family being usually affected. Parents are commonly normal, and females, while transmitting the disease to their male offspring, are as a rule not themselves involved.

Symptoms.—The symptoms develop insidiously. Where the pelvic thigh muscles or the deep extensors of the back are involved early in the course of the disease the gait becomes wobbly and slow, rising is difficult, and climbing stairs still more laborious. The action of the patient on rising from the floor is typical, and once seen never forgotten. The disease may be very widespread or localized, depending upon the type. The proximal movements are mainly affected, the distal less so. The pathological changes occurring in the muscles—whether atrophy, hypertrophy, or pseudohypertrophy predominates—determine their volume and shape, and give a characteristic appearance to each type.

The disease is best described by types:

1. The *pseudohypertrophic*, occurring chiefly in early childhood in the proportion of 3 males to 1 female. Transmission is through the mothers to the males, the parents being free. It involves the pelvis, deep lumbar muscles, thighs, calves, and is characterized by marked pseudohypertrophy in certain muscles—as the deltoids, glutei, and calf muscles—with atrophy of back, abdomen, and upper extremities, marked lumbar lordosis, tilted pelvis, and winged scapulæ.

2. *Juvenile form (Erb)*. This develops at puberty or in middle age, with special and early involvement of the shoulder girdle—weakness, then atrophy, then paralysis. Lower extremities only involved later. Arms more involved than forearms. At times real hypertrophy, or pseudohypertrophy of the deltoid may occur.

3. *Leyden's type*, in which the muscular weakness begins in the pelvic region.

4. *Landouzy-Dejerine or facio-scapular-humeral type*. In this type the atrophy begins in the orbicularis. Then other facial muscles become involved, the facies myopathique develops—the tapir mouth, pouting lips that close badly, fat nose, half-closed lids; later shoulder dystrophy, and finally general involvement ensues. Thorax deformities are commonly present.

Fig. 1



Brothers with Primary Myopathy.
(Montefiore Home.)

PLATE III

Fig. 2



Brothers with Primary Myopathy.
(Montefiore Home.)

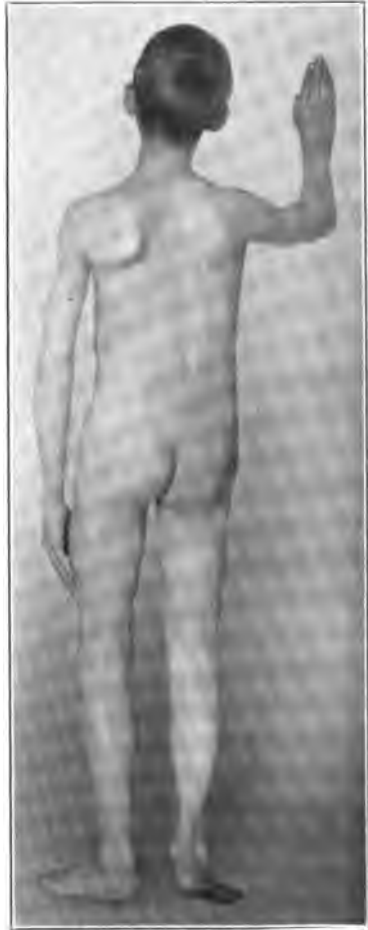
PLATE IV

Fig. 1



Primary Myopathy and Obesity. Note Talipes Equinovarus. (Montefiore Home.)

Fig. 2



Primary Myopathy, Flaring Scapulae. (Montefiore Home.)

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5. *Zimmerlin type*, beginning in the thorax and forearm muscles, the lower extremities being involved only later. This type begins at puberty.

6. *Congenital dystrophy*, a congenital aplasia with secondary dystrophy. Almost all the muscles are involved, including those of the face, eyes, tongue, masticatory muscles, etc. It dates from birth, and bears a superficial resemblance to Little's disease.

The fundamental pathological process underlying muscular dystrophy, and allied diseases, is a diminished developmental power of certain body cells, which leads to a degeneration at puberty. It is possible therefore that a more intensive development may favorably influence these weak elements, and in the discussion on treatment this must be borne in mind. I wish to refer here to what I have previously said under the head of prophylaxis.

Treatment.—While primary myopathy is almost universally regarded as beyond treatment, nevertheless, here and there, throughout the literature, well-authenticated cases of cure exist, and very many can point to cases that have persisted thirty or forty years. Cures have been reported by such reliable observers as Erb and Jendrassik. One thing is apparent to all clinicians, namely, that those who are confined early to chairs do far worse than those in whom locomotion is possible. Where a weakness of the spinal muscles incapacitates the patient, proper braces or supports enabling him to move about are important, and these will do much to arrest the spread of the disease. I have spoken of exercises in the earlier pages of this chapter, and refer the reader to these. It must be borne in mind that no specific rules can be given. Each case must be handled differently, inasmuch as the muscular involvement differs in degree and in localization. Walking chairs, Zander apparatus, exercises in baths, are all very serviceable, the latter especially in young children. Massage and passive movements are very beneficial. Overexertion must be carefully guarded against. Various hydiatic procedures, of a mild and stimulating sort, are useful. Better hygienic surroundings, fresh air, wholesome and nutritious dietary, with special attention to the limitation of fat producers, are also helpful. Mild galvanism, cathodal application, is also beneficial (Oppenheim saw marked improvement in 2 cases under this treatment); but one must avoid treatment by too powerful currents. The use of various therapeutic agents, such as the injection of muscle extracts, with apparent improvement, is reported by Allard and Lordeus. Rossolimo found improvement in a case after using thyroid extract. Thymus and extract of other glands have been employed without distinct benefit.

Where contractures exist, tenotomies have produced marked palliation, rendering locomotion and limited activity, so important in this condition, possible. Tendoplasty and tendon transplantation have also been used with indifferent success, owing to the implanted muscle being already atrophic, or about to become so. In selected cases, I have found transplantation to be of some service. Shortening of the tendons is rarely of service in these conditions.

Various splints may be employed to prevent contractures, and to support parts, especially in shoulder atrophies. Various surgical procedures may be useful, *i. e.*, when the scapulæ are markedly winged, and there is considerable weakness of the muscles which fix the scapulæ, the scapulæ may be sewed together, or else fixed to the underlying parts. Nerve transplantation naturally is not to be considered.

It must be borne in mind that rudimentary and undeveloped forms of dystrophy exist. Here, while the predisposition is present, most likely the exogenous factors did not act as deleteriously owing to greater bodily resistance. These rudimentary forms of the disease should point the way to combat this severe and intractable condition.

Progressive Muscular Atrophy.—We have just discussed the primary form of progressive muscular atrophy. There are two other main forms, and also transitional forms. As in the myopathies the familial character is conspicuous.

The first form is spinal:

(a) The non-hereditary, adult type (Aran-Duchenne).

(b) The hereditary, infantile type (Hoffmann-Werdnig).

Aran-Duchenne Type.—We shall first consider the *Aran-Duchenne—non-hereditary—type*, a disease which rarely occurs before the twentieth year, and usually appears in middle age.

Etiology.—The etiology is obscure, but nevertheless the influence of trauma, of exposure, or of muscular overexertion is very important, especially that of trauma, which gives rise to a distinct type of the disease.

Lues has been repeatedly found in the etiology, and while its direct causative influence has been denied by various authorities, nevertheless more and more cases are appearing in the literature. I have seen a number of cases where syphilis appeared to be the essential etiological factor of progressive muscular atrophy; and an acute poliomyelitis in childhood has been the starting point later in life of a progressive muscular atrophy.

The Aran-Duchenne atrophy is a slowly progressing muscular disease. The muscles first involved are usually the intrinsic hand muscles, the opponens pollicis, and the first interosseus; a flattening soon occurs; certain movements are lost, and the thumb assumes an unusual attitude. The characteristic feature of the disease is that the atrophy is preceded by noticeable weakness. Other symptoms are fibrillary twitching, diminution and loss of deep reflexes, diminished and altered electrical reactions, and even reaction of degeneration may be present. The atrophy is bilateral and fairly symmetrical, with little or no pain. In certain cases the disease begins in the shoulder muscles. There is characteristic flattening of the supra- and infraspinous fossæ, and of the neck and of the cervical spine. Then the deltoids are involved. In the hand type the spread is toward the shoulders, and in the shoulder type toward the hand.

Course.—The course of the disease is gradual—an involvement not by contiguity, but by leaps from the hand direct to the shoulder. There are

PLATE V

Fig. 1



Progressive Spinal Muscular Atrophy. Note Shoulders and Hands. (Montefiore Home.)

Fig. 2



Approximation of Scapulae, due to Muscular Atrophy. (Montefiore Home.)

long remissions sometimes. Sometimes it appears as if the disease had been arrested. I have had such cases under observation for ten years, with no definite advance. Oppenheim doubts these cases, and thinks that they should be classed with chronic anterior poliomyelitis, or with the occupation atrophies. Yet my cases and numerous others reported in literature were neither occupation atrophies nor chronic poliomyelitides. Life is endangered by paralysis of the respiratory muscles. Bulbar paralysis is a very frequent terminal complication.

Pupillary changes have been repeatedly observed, even stiff unequal pupils. These cases may be either luetic, a combination of the disease with tabes, or wholly due to the progressive muscular atrophy.

The disease has its distinct ear-marks, and only rarely is it difficult to make a diagnosis. It is not uncommonly seen complicating locomotor ataxia.

Pathology.—The pathology is, briefly, a progressive degeneration of the ganglion cells in the anterior horns, in which the muscular atrophy is directly proportionate to the change in the anterior horns. The atrophy of the muscles is due to a trophoneurosis, the muscle fiber suffering from a loss of activity and loss of power of assimilation of nutrition. The atrophy has a segmental distribution, and is symmetrical.

General Treatment of Progressive Muscular Atrophy (Spinal).—Medicine has been from the beginning the art of healing; the public who furnish us with the schools of learning, the various laboratories for research, gives us the right to employ our art, and safeguards that right by laws regulating the practice of medicine; this public has in mind the treatment no less than the prevention of disease. The physician who honors the specialist by calling him in consultation, as well as the patient, desires information mainly as to treatment. With modern laboratory methods, and modern teaching, the family physician has already made his diagnosis in the majority of the cases. What he wants to know is how the treatment, barely sketched in the text-books at hand, is to be carried out in the individual case of his own patient, and what is the relative value of the various modes of therapy vaguely mentioned: in short, what has greater experience taught the specialist regarding the most efficacious method of handling the malady of his patient.

There are physicians who boast only of their skill as diagnosticians. When it comes to therapy their suggestions are as barren as those found in the most elementary treatises. Indeed, the great weakness of the modern institution of consulting diagnostician is that for years he has not had the treatment of patients under his immediate charge; for years he has busied himself with seeing patients only for a sufficient time to make a diagnosis. What the result of any of his suggestions as to treatment has been, he rarely learns. The patient very rarely desires to see the same consultant a second time; his knowledge has been exhausted; no doubt another will give newer and better advice.

Of course it will be asserted that the diagnostician obtains his therapeutic knowledge in the various hospitals with which he may

be connected. But this pertains to those in active service, and **every** one knows how little attention is paid, as a rule, by the visiting **physicians** of most hospitals, to the details and the manner of execution of the various general orders given by them. The hospital rounds become mainly the demonstration of the latest or most unusual symptoms **for** the benefit of the resident staff or students.

What I have said appertains to a greater extent in the treatment of chronic disorders; in the vast majority of which, for all concerned, the diagnosis is of lesser value than the therapy.

I do not wish to underrate the value of a correct diagnosis; this **is** self-evident, but it behooves the patient, and the physician more, to know the best management of the case, than to know whether or not the case is a syringomyelia, or a central gliosis, or an amyotrophic lateral sclerosis, or a Marie, or a Friedreich's ataxia. In fact, so far as the family physician and the treatment are concerned, it makes precious little difference what type of myopathy is at issue.

Of course it may be said that the therapy ends with the diagnosis. So far as the treatment of the patient is concerned, it often does. It is especially in progressive muscular atrophy that this view is so frequently advanced. But anyone who has patiently and conscientiously treated this disease knows how much can be done to arrest it for an indefinite period, if not to effect a cure. Every neurologist of experience has had numerous cases in which, after treatment, little or no progress was evident during years and years of observation.

According to some authorities, such a remission is commonly observed in the course of this disease, and must not be attributed to treatment. Yet, according to my experience, and that of other observers, improvement, in steadily progressing cases, does result from treatment. Furthermore, when the treatment is interrupted, over a long period of time, for any cause, the disease again begins to advance, and can be checked once more by treatment.

While this course has not been invariable, it has occurred sufficiently often to warrant painstaking endeavor on the part of the physician.

As to the generalities: Firstly, I desire to refer the reader to that part of the chapter which deals with muscular atrophy in general, and the causes thereof, and to the treatment mentioned therein; the lesson contained is one that must be borne in mind in the treatment of all atrophic states. Each case must be handled according to its etiology.

The cases presenting a definite luetic history must be handled like any other syphilitic disease of the nervous system. Where there has been insufficient treatment for the syphilis, an antiluetic treatment ought to be instituted. Salvarsan in repeated small doses, 0.3 gram every second week for two months, then mercurial treatment for one or two courses of hypodermic injections of bichloride, or salicylate, or enesol. The doses should not be maximum doses; deep injections, gr. $\frac{1}{2}$ of sublimate twice weekly, gr. 1 of salicylate once a week, or $\frac{1}{2}$ ampoule of enesol twice weekly. Should improvement be definite, then an intermission can be allowed, during which the treatment about to be

mentioned can be carried out. If a marked augmentation of the weakness or wasting occurs, owing to the possible deleterious effect of the arsenic, or the mercury on the lower motor neurones, then this form of therapy must be discontinued, just as must sometimes be done in the treatment of tabes and general paresis.

If one is dealing with a definite traumatic progressive muscular atrophy, the patient must be taken away from occupations where traumata are likely to occur. If seen within a reasonable time after the trauma, and if the atrophy is extensive but localized to the injury, then a rest in bed for a few weeks, moderate and not exhausting massage, or various other mechanical treatments accessible, ought to be applied. Rest, vibratory massage, and electrical massage are useful; also mild faradism, cathodal galvanism to the muscle, anode to the spine. I have empirically applied, once weekly, a counterirritant to the spine, over the region of the segments supplying the affected muscles—mild irritants, such as mustard, cantharides, even the Paquelin cautery. Scotch douches, mild spinal douches, warm baths with exercises while in the water, are also advisable.

While the patient is in bed, a nutritious diet, easily assimilated, should be given, although no special restriction of the class of food should be made, a low purin diet is preferable.

As to drugs—strychnine should be given hypodermically, according to rules about to be detailed; local administration is preferable.

In more advanced cases, the duration of the rest in bed will depend on the rapidity of the atrophy. If rapid, rest is imperative; if very slow, adequate rest to inaugurate a systematic therapy is necessary. Once the treatment is started, the patient remains at his best when kept moderately active. Excess or overactivity is deleterious.

For those cases with unknown etiology the treatment again depends on the stage of the disease. In early, and in slowly progressive cases, where the atrophy is localized to one or both hands, or shoulders, and is not very extensive, the treatment outlined above for similar cases is advisable.

In these cases, the intensive strychnine treatment must be instituted at once. Given hypodermically, strychnine is of great benefit, beginning with gr. $\frac{1}{80}$ or even gr. $\frac{1}{120}$, daily or at least three times weekly, near the site of the atrophy. The dose is slowly and steadily increased. A careful watch for trismus, or tremor, or other evidence of strychnine intoxication is kept. The dose should remain close to this boundary line; occasionally it must be increased to define the upper margin. My observation has been that, with time, most patients acquire an increasing tolerance for the drug, and I have often given as high as gr. $\frac{3}{4}$ in a single dose to patients who have been taking the drug for a very long time. The increase must be carefully graduated, and the patient sedulously watched. I have never observed dangerous symptoms, and where trismus and muscular overexcitability have occurred, absolute rest in bed and quiet sufficed in a short time to remove the untoward symptoms. If by mistake an overdose has been given, then treatment

for strychnine poisoning must be instituted. The physician bearing in mind the frequency of the injections, must employ strict antisepsis; iodine, over the site of injection has proved valuable. The amount of fluid injected should be the least possible compatible with accuracy. In the solutions employed five minims should equal gr. $\frac{1}{60}$ until large doses are required; then more concentrated solutions are necessary. The physician should not intrust this solution to anyone. I have seen a nurse mistake it for the stock solution for internal administration, with unpleasant consequences.

For the rapidly progressive cases, rest in bed is imperative; the other treatment is the same. The results are not as grateful as in the less rapid types; nevertheless, improvement is not by any means uncommon.

Thyroid medication, increasing local muscular metabolism, is to my mind contraindicated, and I have never seen the least benefit therefrom. Arsenic in combination with iron and strychnine may be used during the remissions. I have employed arsenical preparations hypodermically, yet the results in the vast majority of cases have not been as gratifying as with strychnine. In advanced cases, with considerable atrophy and with flail-joints, various orthopedic appliances are very useful. In dropped wrists and dropped fingers a glove-like apparatus has enabled my patients to use their hands much more effectually. In a similar fashion a shoulder brace has overcome some of the disability due to an atrophied deltoid, with subluxation of the humerus. A skilled orthopedic surgeon is able to overcome many of the disabilities and render possible a considerable amount of exercise by the patient himself.

For flaring and winged scapulæ, fixation may be done.

Tendon shortening may be employed in very slowly progressive cases.

Nerve anastomosis is rarely employed, owing to the probable involvement of the centres of the nerves employed.

Swedish movements and Zander apparatus methodically and moderately employed, I have found useful to supplement massage.

Tobacco and excessive alcoholic indulgence should be interdicted.

Favorable climatic surroundings act as a general tonic.

Before closing, I desire to call attention to institution treatment. In fairly advanced cases, and in early cases, where there is a justifiable suspicion that treatment will not be thoroughly followed at home, cases of progressive muscular atrophy ought to be sent to institutions adapted to the treatment of chronic nervous diseases. My results at the Montefiore Home, a hospital for chronic diseases, have led me to prefer this method of treatment to the best given at home. Zander, hydrotherapeutic, and electrical apparatus, with internes and nurses trained and adapted to the requirements of this therapy, with the local regime, the constant presence of others benefited by the same treatment, all tend to encourage the invalid, and to make him contented under the long and trying cure.

Above all, patience on the part of both invalid and physician is requisite, and slowness of progress should not discourage either.

Progressive Muscular Atrophy of Infants and Children.—There are three main types of this atrophy:

1. The Hoffmann-Werdnig type, which occurs only early in life—in the first days or first months—and terminates after a varying period in death. It is familial in character. The infant has a complete flaccid paralysis of all four extremities, the distal parts being especially involved. The muscles are atonic and flaccid. The atrophy is masked by subcutaneous fat. The sensorium is uninvolved and sensation intact. The tendon reflexes are diminished or absent; reaction of degeneration is present.

The pathology consists of very marked changes either in the motor fibers, in their intra- and extramedullary course, or of the ganglion cells themselves, or both; slight variations depend upon which site is the more involved. The picture resembles an intense intoxication plus an abiotrophy.

2. The second type is similar to toxic neuritis, and begins somewhat later in life—after the child has walked. It progresses slowly, terminating in death from respiratory embarrassment.

The pathology here is a toxic degeneration of the lower motor neurone. The onset is rather sudden; all deep reflexes disappear, incontinence of urine and feces is present, reaction of degeneration develops, and the distribution of the paralysis is more distal than in the first type.

3. The third type also begins after the child has walked, and progresses steadily.

The pathology is an extensive chronic myelitis, with atrophy of the anterior lower horns. The disease ends in death. The picture is that of a chronic diffuse myelitis.

The treatment of the above conditions is merely palliative.

Chronic anterior poliomyelitis bears many points of resemblance to progressive muscular atrophy, and is at times difficult to differentiate from it.

A gradual weakness develops in one or other extremity; first a lower, next an upper, or the other lower, and then the other extremities are affected, until after a few months there exists a total paralysis of all four. The paralysis picks out certain muscles, and spares others with no regularity, and no symmetry, and with usually no tendency to distal involvement. A flaccid, atonic paralysis, with abolition of deep reflexes, and with reaction of degeneration, and fibrillation develops. Paralysis and atony precede the atrophy. Sensation remains normal.

The course of the disease may be stationary, remitting, or intermitting, and ending in death.

The treatment is similar to that of progressive muscular atrophy.

The Neuritic Type of Progressive Muscular Atrophy (Peroneal or Charcot-Marie Tooth Variety).—This begins in the second half of childhood, or later, even to the third or fourth decade, and is familial

in character. It is usually directly transmitted, but it may skip a generation. Sherrington has reported twenty-six cases in one family.

The onset is slow, atrophy being in the muscles of the foot, chiefly the peroneals, extensor communis, digitorum, and intrinsic foot muscles. Deformity of the foot soon develops—club-foot, varus or equinovarus, claw toes, and ankylosis or flail-joint. Later, the gastrocnemii are involved. After years, the upper extremities are affected. The thenar or hypothenar regions and interossei are attacked, claw hands develop. The spread is essentially distal. Fine tremor and fibrillation are present, and an incomplete reaction of degeneration. Limited tenderness of nerve trunks is the rule, the knee-jerks disappear, and slight subjective and objective sensory disturbances in the peripheral portions of the limbs develop. Trophic disturbances are rare.

The treatment is orthopedic. Its results at times are fairly successful in diminishing the discomfort of the disease. For further details see treatment of myopathies.

Progressive Bulbar Paralysis.—There are two forms of Progressive Bulbar Paralysis: an extremely rare infantile hereditary form (described by Fazio and Londe) resulting in death, and a type occurring rarely before fifty.

This latter shows a slowly developing difficulty of speech, swallowing, mastication, and phonation, which arises from and is dependent upon a symmetrical paralysis and atrophy of the lips, tongue, palate, pharynx, larynx, and muscles of mastication.

The speech difficulty usually occurs first, and the patient becomes easily fatigued by continued talking. The speech becomes more indistinct. At first the linguals, later the labials, are affected and then nasal speech develops. Soon dysarthria, then dysphagia, regurgitation of fluids through the nose, coughing spells during swallowing appear. Next, inability to swallow develops, then mastication is lost, phonation disappears, and lastly respiratory difficulties develop.

The pathology is a progressive degeneration of the nuclei of the medulla and pons, with secondary atrophy, and paralysis of the parts supplied by the nuclei.

Atrophy, fibrillation, and partial reaction of degeneration are found. The patient becomes very emotional. The facial expression is characterized by open mouth, hanging lower lip, drooling, lower half of the face expressionless. Rapid emaciation occurs later. The progress of the disease is very slow.

The therapy is symptomatic, and efforts must be made to sustain the vitality. Such drugs as silver nitrate, strychnine, potassium, arsenic, in conjunction with careful feeding, and galvanism, may be employed.

The infantile type is an hereditary, familial disease, with such marked stigmata of degeneration as prognathism. It begins with paralysis of the facial muscles, ophthalmoplegia and lagophthalmos; then bulbar symptoms develop; it is characterized by remissions, and exacerbations, muscle paralyzes and atrophy, with partial reaction of degeneration.

PLATE VI

Fig. 1



Chronic Anterior Poliomyelitis. Note the wasting of the muscles of the left scapula and arm. (Montefiore Home.)

Fig. 2



Progressive Bulbar Palsy. Note tongue atrophy and the overaction of the facial muscles. (Montefiore Home.)

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PLATE VII



Amyotrophic Lateral Sclerosis with Bulbar Symptoms.
Note bulbar facies. (Montefiore Home.)

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It is closely related to the infantile nuclear disappearance of Heubner.

Treatment is the same as that just above described.

Amyotrophic Lateral Sclerosis.—This is a disease of middle life. It is rarely found in children, and is rarely of familial nature.

Etiology.—The etiology is little known, but in the various histories exposure to cold and trauma frequently stand in an apparently causal relation. Strümpel believes that we are dealing with an endogenous weakness of the motor apparatus, which, with the addition of the exogenous factors in later life mentioned above, produce the disease. The frequent occurrence of micrognathia and congenital deformities of the thumb seem to bear this out.

Pathological.—The pathological anatomy shows a disease, intensive in character, of the pyramidal tracts, and the ganglion cells in the anterior horns and medulla, with a characteristic preservation of the tracts containing the reflex collaterals. Briefly it is a disease of the upper motor neurone, which has been traced in some cases to the Rolandic cortex, and the ganglion cells of the medulla and cord.

Symptoms.—It follows that the clinical picture is that of a spastic paraplegia, plus chronic poliomyelitis of the cord and bulb. It is a chronic progressive disease in which for a time either clinical picture may predominate, or both may run apparently in parallel fashion. In some cases, for a long time the disease may appear as a spastic paraplegia and only later a muscular atrophy develops; or else it may resemble a progressive muscular atrophy, and the later spastic phenomena may appear either in the lower or in the upper extremities, with rigidity, exaggeration of the tendon reflexes, patellar and Achilles clonus, Babinski, etc., in the lower extremities. Pain and paresthesia are naturally uncommon. In the terminal state, as in progressive muscular atrophy of spinal origin, progressive bulbar paralysis supervenes. The duration of the illness is very much briefer than in the former case, death supervening in a few years, at the latest, in the vast majority of cases.

Treatment.—The treatment, in view of the very bad prognosis, is naturally limited to sustaining the patient, and to the amelioration of the individual symptoms.

For the treatment of the muscular atrophy and the bulbar symptoms, the reader is referred to the sections on progressive spinal atrophy and bulbar paralysees respectively.

For the treatment of the spasms and rigidity, as the disease is eminently progressive, and of fairly rapid character, very little of lasting good can be done. Tenotomies may be useful for a time. The question of posterior root section is rarely to be considered, and then only as a palliative treatment. Yet experience has taught us the futility of this procedure in progressive conditions, and considering the dangers of the operation in the hands of any but the most experienced spinal cord surgeons, it ought to be very rarely considered, and only in slowly progressive cases, without any bulbar implication

Strychnine, given hypodermically, for the atrophy, prolonged warm baths, 95° F. for twenty minutes to an hour for the spasticity, may be usefully employed.

Myotonia Congenita (Thomsen's Disease).—I have interpolated this disease, in order to make the study of myotonia atrophica, which follows next, easier.

Myotonia congenita was first described by Bell in 1832, but it was not until 1876 that Thomsen called general attention to this peculiar malady by giving a complete description of the cases occurring in his own family. Later, Strümpell named it myotonia congenita, and it remained for Erb—in 1886 to 1889—to give the best monograph on the subject up to that time.

Etiology.—It is a congenital and familial disease, occurring commonly in childhood, but which may appear at a much later date. In the latter case, exogenous factors, such as fright and emotional shock, seem to play the part of immediate causes. The great preponderance of males is noteworthy.

Pathology.—The pathogenesis is still *sub judice*. The earliest observers believed it to be an affection of the nervous system; as a defective or diminished influence of the will or nervous system on the muscles. Later investigators, studying the ergographic tracings, and bearing in mind the muscular changes, and local mechanical irritability, believed it to be a disease of the muscular end plates, of congenital origin. Still later investigators found metabolic changes, and believed that the accumulation of toxins in the muscles produced the clinical symptoms, and the pathological changes. They also pointed to the existence of acquired myotonia.

Jensen showed a faulty muscular assimilation, and diminished excretion of carbon dioxide, lactic acid, and xanthin bodies. As mentioned before, in the general study of hereditary conditions, two factors probably exist; an endogenous irritable weakness of the sarcoplasm, and an exogenous factor of metabolic origin, determining the time and site of the occurrence.

Symptoms.—Myotonia is the characteristic symptom. This consists of a tonic spasm, occurring in muscles long at rest. When suddenly thrown into powerful contractions, the muscle remains contracted for a varying time—from ten to thirty seconds—during which time no effort of the will can overcome the contraction; a gradual relaxation then takes place. Under the influence of frequent repetitions, at brief intervals, this myotonic contraction does not occur. In this manner long-continued movements, such as dancing, walking, etc., are well executed. The spasms may be local or general, the arm may be left outstretched, or the first clenched. Reflexly in the course of coughing or sneezing, or owing to local or mechanical irritation, a remote portion or the entire body may become rigid. This must be borne in mind in the treatment of this condition.

In affected families the disease may be noted in early life. As Strümpell has called attention to the defective congenital functioning

of the facial muscles often seen in the children of dystrophic families in which they sleep with open eyes, never learn to whistle, etc., so in myotonic families, the children may show in early life difficulty in sucking, in opening the eyes, an immobilization of the face during weeping, and a certain clumsiness in getting about. The awkwardness of such children is most frequently attributed to their heaviness.

Patients with myotonia are individuals of excessive muscular development, local or general; they look like the individuals seen on the frontispieces of various athletic journals.

The affection may be localized in the facial, masticatory, or ocular muscles or tongue, or may be general. The myotonia is increased by exposure to cold, by emotional excitement, by steady observation, or upon sudden intention. Conversely, peace of mind, moderate stimulation by alcohol or tea, and methodical exercises tend to diminish it.

Erb has called attention to the following pathological signs:

1. The increased mechanical irritability of the muscles and not the nerves; a slow tonic contraction of the muscle follows a local tap.

2. The myotonic electrical reactions. Constant weak faradic stimulation of the nerves is normal; constant powerful currents produce contractions of prolonged duration; interrupted currents only cause brief contractions. Direct muscular faradic irritability is increased; even feeble currents cause prolonged contractions; with constant currents, undulatory movements in the muscle can be seen. The galvanic nerve reaction is diminished; only interrupted currents cause contractions; direct muscular galvanism is augmented; only closure contractions occur; usually ACC = or > CCC; the contractions are sluggish, tonic, and of long duration; undulation is also observed with stable applications; the contractions pass from the anode to the cathode; a strong current is necessary to elicit the phenomenon.

Irritability to static currents is normal.

The myographic curves with Mosso's ergograph are characteristic. The intensity of the disease varies considerably.

Other distinctive signs are absent, except that various observers have found in the urine excess of uric acid, of chlorides, of tyrosin, and of xanthin bodies. Glucose has also been found in some cases.

Myotonia appears in numerous affections, such as paramyotonia congenita (Eulenberg); myotonia congenita intermittens, myotonia congenita acquisita and transitory (Jacoby); myotonia acquisita (Talma); ataxic myotonia (Gowers); myotonia with myoclonia (Lundberg).

Course.—The course of the disease is chronic and permanent; beginning generally in early childhood, increasing up to the third decade, and then remaining stationary. Remissions and exacerbations are not uncommon. Prognosis as to life is good; as to recovery is poor.

Treatment.—The therapy, in view of the incurability of this affection, resolves itself into a symptomatic one. Nevertheless, various attempts have been made to influence the disease. For a time orchitic extract and thyroid extract were employed with indifferent success; atropine and strychnine were similarly found inefficacious. From massage and

gymnastics much good will result, so far as relief is concerned. Local or general massage, Swedish movements, and Zander apparatus ought to be employed. In a similar way electrical massage may be serviceable. For details of this treatment, *vide supra*.

Prolonged warm baths of 95° F. are available in general involvement. They conduce to diminish the tendency to rigidity. In fact, as mentioned before, warm baths are well borne and well liked by the patients.

Other drugs such as iodide of potash and antipyrine have been employed in vain.

For the early cases the reader is referred to the section on Prophylaxis, where the methodical treatment of hereditary diseases is considered, and the following of the precepts mentioned therein will do much to retard, if not check, the further development of this severe disorder.

Myotonia Atrophica.—The characteristic features of this affection are weakness of the facial musculature, atrophy of the sternocleidomastoid muscle, of the vasti of the thighs, and the dorsiflexors of the foot, and the slow relaxation of the various muscles after contraction (the myotonia). The more rapid and forcible the contraction the slower the relaxation.

Various synonyms are Thomsen's disease with muscular atrophy, atypical forms of Thomsen's disease, muscular atrophy with electrical reaction of Thomsen's disease, myotonia and muscular atrophy. The disease is an uncommon one and has a familial character.

Etiology.—The etiology is unknown, but here again exposure to cold, acute rheumatism, etc., plays the part of immediate cause. The disease affects males chiefly, and is a disease of early adult life. The myotonia commonly precedes the atrophy.

Symptoms.—The muscles that atrophy are the facial, the masseters and temporals, the sternomastoids, the trapezii, the flexors, and to a less extent the extensors of the forearm, the vastus internus and externus, the anterior and posterior tibial, and the laryngeal muscles. Other muscles are but rarely affected.

The myotonia is seen chiefly in relaxing the grasp; other muscles show it, but not so constantly. It bears no relation to the atrophy.

The disease is chronic and steadily progressive, and terminates in death.

Treatment.—The treatment is like that for progressive muscular atrophy.

PLATE VIII



Myatonia Atrophica.

**Note dropped feet, atrophy of hand muscles, and facies.
(Montefiore Home.)**

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CHAPTER IV

THE TREATMENT OF HEADACHES

By SMITH ELY JELLIFFE, M.D.

FOR years headache has been considered an entity, although the keen penetration of the early Greek scholars of medicine established the fact that some headaches were symptomatic. Thus it was known that alcoholism resulted in headache. Hippocrates dwelt upon the headaches due to epilepsy, and it is not at all certain but that the headache of migraine was recognized as a separate feature, although in the works of Hippocrates himself we find no evidence to support the belief that he knew of it as an entity.

With advancing years, however, the relationships of headaches to other bodily disturbances have become more and more firmly fixed, and at the present time, deliberately jumping over all of the nosologies of the past, headache is practically regarded solely as a symptom, and not as any disease *per se*.

Any treatment, therefore, of headache, or of pains in the head, means fundamentally a diagnosis of the cause, and the application of measures directed to the removal or modification of such causes.

It will be recalled, however, that this very definite statement regarding the causal nature of headache is of comparatively recent formulation, for should we trace the history of the treatment of headache only back say one hundred years or so we would find that eminent clinician Cullen writing that "headache as a disease is obscure, as a symptom difficult. It may be allowed to be generally symptomatic, but I presume it may also be primary, and much confusion has arisen in the attempt to distinguish them."

It would be difficult to understand what is meant by primary and what by secondary headaches, for after all pain is a cerebral function, a combined function of the thalamus and of the cortex, so that, to a certain extent, all headaches may be said to be primary. But, should we fall in with Cullen's spirit and endeavor for the sake of convenience to indicate divergent lines of interpretation, we can retain his notion of primary headaches and of secondary headaches; primary headaches being those originating within the brain structures themselves, and secondary headaches are those originating in other organs of the body, secondarily involving nervous tissues.

However satisfactory this may seem to be it lacks precision, however, for after all a headache, for instance, from uremic poisoning, which one may say is primarily a kidney disease, and secondarily a headache, is

a toxemia within the brain structures. Just how one would choose to regard the conversion headaches of hysteria, whether primary, secondary, or what not, along such lines of classification it would be difficult to conceive.

We, therefore, choose to reject all such categories as primary or secondary, or functional or organic, and purpose to enumerate some of the more important classes of headaches, noting at the same time some of the clinical features which are of service in demonstrating their closest etiological alliances, providing thereby a rational mode of approach to the question of treatment.

For this purpose, it may be of distinct service to consider those headaches which are more distinctly due to neuralgic processes. These are eminently neuralgias of the fifth nerve, although this is by no means the only nerve involved.

Contrary to many teachings, it is not true that the cerebral structures themselves have no sensibility. It has been taught for years that the viscera have no sensibility, and while it is true that contact with the fingers, with a sharp instrument, by pinching, by the application of heat and cold, etc., have been shown to be unresponded to by the sensory structures of the internal organs, notably the liver, the intestines, the spleen, etc., nevertheless all that this proves is that these forms of stimuli were not of an adequate type; in other words, these structures do not respond to these types of stimuli, any more than the eye responds to sound stimuli, or the skin to taste stimuli. It has been demonstrated beyond a doubt that the viscera do respond to a special form of stimulus, namely, the stimulus of deep pressure, as outlined by Head, and it is this type of sensibility that is also present in the cerebrum, and which is responsible for many headaches. The pain of deep pressure sensibility occurs in all those types of headache associated with increased intracranial tension, whether it be due to the pressure of a new growth, to an abscess, to a hydrocephalus, or pressure from without.

These are not, as a rule, headaches which can be interpreted as due to trigeminal involvements in the narrow sense, although it is not at all impossible that many of the stimuli of deep pressure sensibility may pass through the trigeminus, to the proper perceptive organs. The exact paths traversed by deep pressure sensibility of this type is by no means known as yet in view of the complexity of the surfaces, internal and external, of the cerebral structures. It is for this reason that, for the time being at least, we would exclude intracerebral tension pains from the neuralgic pains that are to make up the first section of this discussion.

EXTRACEREBRAL PAINS

Pains about the Head.—These properly need to be excluded in a differential diagnosis and any rational therapy. A large number of these pains are known, and are loosely grouped with the headaches.

As has already been stated, these, for the most part, are neuralgias, occasionally myalgias, not infrequently the muscular pains of occupation neuroses, and the pains of periosteal or bony inflammatory disease.

Neuralgias.—The most persistent neuralgias of the face are those of the trigeminus. But *tic douloureux*, in its ordinary sense, is rarely confused with a headache. This is particularly true when the two lower branches are involved, but is not so apparent when the supra-orbital is the site of the pain. When this latter branch becomes involved, a diagnosis of headache is not at all infrequent and it is imperative at times to separate such a form of neuralgic pains from a neuralgic pain due to migraine, or to a brain tumor, to a pachymeningitis, a history of neurasthenia, or an eye muscle occupation neurosis. All these types of headache pains in or about the supra-orbital region may be confused with a supra-orbital, *i. e.*, fifth nerve neuralgia.

The first step in the differential diagnosis consists in the accurate plotting of the area involved in the pain. This should be done by methods of testing as outlined by Head and Holmes (*Brain*, 1912), and an esthesiometer is desirable in plotting the topography involved. The location is usually frontal, extending to the top of the cranium; it is, as a rule, unilateral, the base of the nose, the upper eyelid, the ethmoid region deep within the nasal region are the chief sites of the pain. It usually has the characteristic jumping or shooting-like features of a neuralgia, and is, as a rule, accompanied by tenderness to pressure along the nerve trunks. Thus the supra-orbital foramen shows an exquisitely tender point, the ethmoid points are tender, the parietal tubercle, and the inner angle of the eye all show tender points to pressure, indicating a neuralgic process in and along this nerve.

The most frequent causes for supra-orbital neuralgia, as an isolated occurrence, are postinfluenzal and postmalarial toxemias. These carry within themselves their proper therapeutic suggestions. The postinfluenzal neuralgias are helped by aspirin and by iron, whereas quinine, arsenic, and iron are the best modes of therapy for the postmalarial supra-orbital neuralgias.

Occasionally intestinal parasites are known to produce a neuralgia of this nerve. Leukemia and diabetes may infrequently be the cause. In any of these instances the etiological therapy is clearly indicated.

A true Gasserian ganglion disturbance, involving the supra-orbital branch alone, is comparatively infrequent, yet occasionally such cases have been met with. Here, however, as a rule, other branches are occasionally involved even though the supra-orbital branch is that most persistently affected. In such instances, after careful etiological deductions have excluded all other forms of possible causation for the supra-orbital neuralgia, surgery or alcohol injections may be used, in the event that the pain is severe and intractable. In this case, however, the relationship to headache is more nominal than real; it is evidently a supra-orbital neuralgia of definite etiological causation.

Affections of the middle and inferior branches rarely cause headaches, although occasionally involvement of a dental branch will cause

a temporal headache. This has been known to take place with carious teeth, and the temporal headaches which have resulted have been very refractory to all forms of treatment.

Occipital Neuralgias.—The occipital neuralgic headaches offer an equally fertile field for differential diagnosis. Like the pains of supra-orbital neuralgia the occipital neuralgias are due to the involvement of special nerve fibers. Here the occipitalis major, occipitalis minor, or the auricularis magnus are those most frequently involved. Here, again, intracerebral complications causative of headaches are to be separated from those of purely external origin. Most frequent of these for instance are caries of the vertebræ, tumors high up in the cervical cord, and occasionally posterior fossa pachymeningitis, or cervical meningitis. The occipital neuralgias furthermore are found in syphilitic affections. These constitute a very important group of the occipital neuralgias, and often are unrecognized for long periods of time. All of the occipital neuralgias, especially when mild, are frequently misinterpreted as occipital headache, and are miscalled "neurasthenic."

They are furthermore confused with a very common form of occipital pain which is an occupation neurosis, and also with the pains due to indurative myositis which is especially prevalent in the occipital region.

Occipital neuralgic headaches are almost invariably bilateral, and when true neuralgias almost invariably are associated with painful Valleix points, which may be found along the cervical outlines. When the pains are sharp in character they are obvious. They then stamp themselves as neuralgias. But they are frequently dull, and are increased or are brought about by movements of the head. They frequently reach the vertex, and it is significant of this type of pain, if due to neuralgia, to find occasional twinges of pain in the shoulders, running down the arms, over the scapular region. In brachial plexus cases, where there is very slight involvement of the nerves of the arm, and greater involvement of the recurring branches from the upper cords of the plexus one occasionally meets with sore points in the neck, and also with slightly swollen and tender nerve trunks.

Treatment.—Therapeutic indications are with counterirritation, mild rubefacients, mustard, cantharides, menthol, etc., which should be applied along the Valleix points, or over the swollen nerve trunk in the neck, or over the tender nerve trunks in the scapula. Mild massage is of service, and mixtures containing salicylic acid or anti-pyrine, or other analgesics are indicated.

Occasionally one encounters foreign bodies pressing upon the nerve trunks, in which case their removal is obviously indicated. Syphilitic osteitis may be the starting point of such a pressure, in which case a Wassermann reaction would be utilized in making a diagnosis and the proper therapy instituted.

Many of these posterior occipital headaches of neuralgic origin are present in the chronic stages of malarial poisoning. They are not encountered so often at the present time as formerly, since the discovery of the malarial organism has permitted a prompter therapy of malaria

in general, and in large measure tended to do away with the many forms of malarial cachexia. Such chronic malarial cachexiæ should be borne in mind as a possible cause of a chronic occipital neuralgic headache.

Quinine is here of less value than arsenic, as a rule, although it can be given in doses of from one to two grams.

The anemia that accompanies chronic malarial cachexia, as well as that due to parasites or diseases of the blood-making organs, is best treated by iron and arsenic. Special antineuralgic drugs are to be used only in the more severe cases, and only after a thoroughly consistent diagnosis has been established. The most important of these drugs are antipyrine, phenetidine, phenacetin, salipyrine, aspirin, pyramidon, and other combinations usually of the salicylates. These may be used singly or in combination, in doses of from 5 to 15 grains, according to more obvious indications.

The more powerful of these analgesics should never be administered in more than 5 grains (0.3 gram) as an initial dose even to an adult, especially if the adult is unknown. It is of interest to know that whereas most of the analgesics are pharmacologically related and from the standpoint of chemical structure should have practically the same action, nevertheless, clinical experience shows that the minor modifications in chemical structure are oftentimes reacted to idiosyncratically by different individuals. It is, therefore, well worth while to study these analgesics with discrimination. One can establish for the patient an ideal combination, and one can also play the changes with different ones and thus help effectually to prevent an opium habit in some of the more chronic of these headaches.

While on the subject of the opium habit, it should not be forgotten that these neuralgic pains may be symptomatic of such a habit, particularly during an attempt made to break it. The habitué who contracts a habit as a result of his efforts to relieve pain of this type is very apt to find that the first attempts at suppression of the opium will bring about pain in this same region.

In some of the more obstinate types the older remedies, such as gelsemium, aconite, atropine, and cannabis, may be utilized. Usually, however, they serve only as surrogates to other more direct and profitable therapeutics.

Electricity is of service in a number of these cases, especially in the form of the violet ray, or the high-frequency static spark or in the rapidly alternating current of Leduc; five to ten minutes are usually sufficient. The milder forms appreciate the galvanic and the faradic streams as adjuvants to other forms of treatment. The streams should not be over one to one and a half milliampères, and the length of time rarely over fifteen minutes.

Massage is frequently of value, but it is of less value in the neuralgic type of headaches than in others about to be considered.

Reflex Tenderness of the Scalp.—Ever since Head called attention to the fact that there is an intimate association between the internal

viscera and the skin a rational explanation has been afforded for what has empirically been recognized for several years, namely, that certain disorders of the viscera are frequently, if not invariably, associated with reflex pain distributed in the area of the scalp, or are responsible for tenderness in the skin of the head.

These reflex pains, and this scalp tenderness, are referred to by patients as headaches, and therefore become legitimate subjects for consideration in this chapter.

Later studies by Head himself, by Hilton, Janet, and by MacKenzie, have served to accentuate and bring into sharp relief the anatomical relations, through collaterals in the cerebrospinal axis, that these affected skin areas bear to the affected viscera.

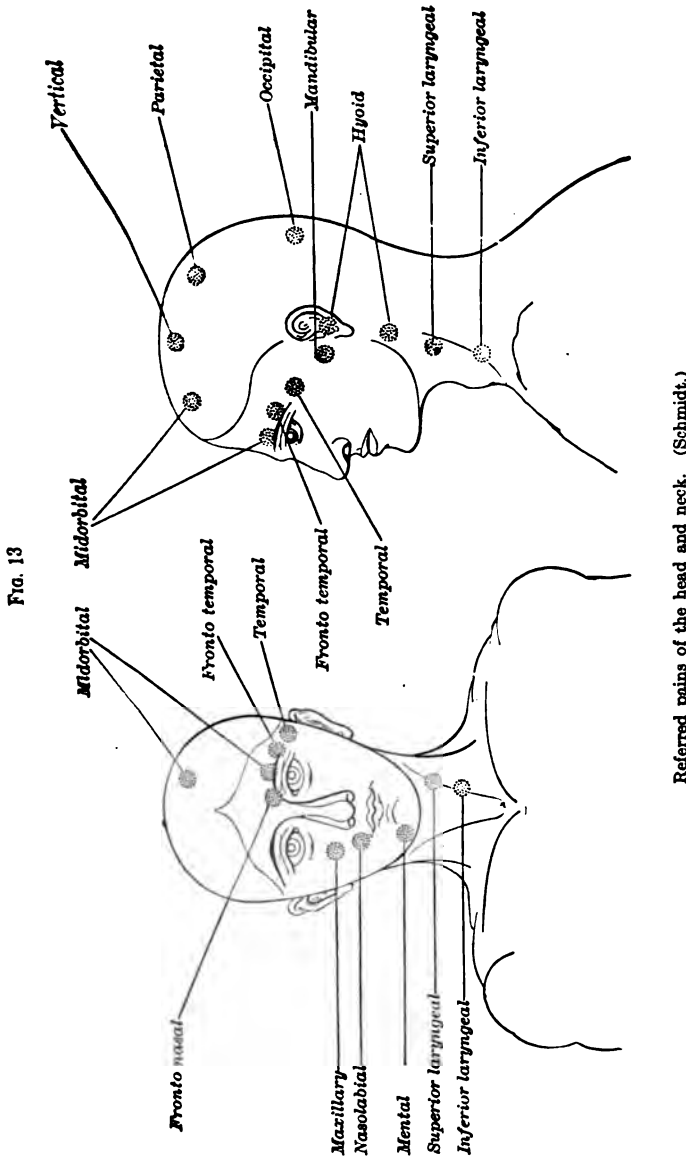
The therapeutic mode of attack, however, must concern itself more with the diseased organ than with the reflex disturbances in the scalp itself. As a matter of clinical experience it is known that the intensity of the hyperesthesia in these areas varies widely, and although it may bear some proportionate ratio to the severity of the underlying visceral disturbance, such a ratio is difficult to express. Not infrequently a comparatively mild visceral disturbance will excite or create a very active reflex; whereas, on the other hand, a very severe internal disease may give rise to comparatively little reflex disturbance in the scalp areas.

It is characteristic of these reflex pains that examination by the rounded glass head of a sharp pin, with von Frey's hairs, or with other esthesiometers, shows that topographically they are not related to any peripheral nerve distribution. In other words, they do not conform to the ordinary neurological types. They are usually much less circumscribed, are often bilateral, and may be extremely fleeting. Patients complain of them as dull aches, as casque-like bands, as sore spots, as tender spots, spots that hurt when the hair is brushed or combed, spots that when touched are found to be tender, and the touch is followed by a persistent dull and annoying ache. The localization of these reflex areas is indicated in the charts, and also some guide is there afforded as to the visceral diseases. These reflex pains must be distinguished carefully from the hallucinatory pains of dementia præcox or a mild manic-depressive psychosis—so-called "hypochondrias."

Treatment.—The therapy requires a twofold mode of approach—the correction of the underlying visceral disturbance and the alleviation of the local discomfort. This type of reflex pain is best relieved by treatment of the disease in the viscera. Locally these pains can often be relieved by hot applications and by rubefacient analgesics. Menthol is one of the most serviceable. Rubbing of the scalp is often of much service in this type of reflex head pains.

Muscle Neuroses.—Another type of headache little understood, but frequently found, is that which may properly be spoken of as an occupation neurosis in certain of the muscles of the neck and scalp. As is well known, many of these muscles are no longer functionally active. Thus the modern man has no use for the muscles of his ears, nor are those of the scalp very much in use; whereas it is not infre-

quent that the muscles of expression, speech, mimicry, emotion are constantly employed, and as for the muscles of the back of the neck, these are nearly always in use.



These types of headache are the result of continuous, and yet for the most part instinctive and unconscious muscular activities. They are muscular activities which consist of a continuous series of automatic

motor adjustments, easily set in operation, for instance, by necessities arising from defects in the chief sensory organs of the head.

Here are grouped the muscle pains so frequently spoken of as eye-strains, ear-strains, neck-strains, and position-strains, because minor defects in the eyes, the ears, the position of the body, etc., call for a continuous functioning of particular groups of muscles, with the result that they become fatigued, and exhibit the symptoms of an occupation neurosis.

Symptoms.—Those headaches which are the most frequent are frontal and occipital. They are often persistent and are characterized by certain features which must be grasped in order that their real essence may be perceived, and efficient therapy be made possible. Most of these headaches come on more or less gradually. The patient observes them after carrying on a more or less definite occupation. After a certain length of time it becomes apparent that this particular occupation is pursued with more discomfort than others, the attention is apt to wander, there is less enjoyment, and finally one notes that for some reason or other one has a headache. This headache is then apt to increase in intensity and in duration, with the recurrence of the occupational strain, and which has perhaps developed only after the individual has been at work for several hours. The head distress becomes more and more intense after perhaps years, and develops in later years sooner, in point of time, after the actual starting of the occupation.

Diagnosis.—It requires, at times, very detailed anamnestic investigation to establish these relations, and if such careful detailed history fails to show such relations it may be accepted that the headache does not fall within that category. It is not enough to find an eye or ear anomaly; the character of the headache is of importance.

So far as statistics are able to inform us, it would appear that eye headaches are the commonest. This is as one might expect, since, other things being equal, there is probably no series of muscles in the body used so widely and persistently as those governing the eye movements, and probably no organ of the body that shows more minor anomalies of structure than the eye. These minor anomalies of structure, combined with the necessity for constant use, bring about a disharmony, and therefore irregularity of tension requiring compensation, with the production of an eye-strain headache. It is very far from being a fact that every anomaly of structure produces headaches of this kind, any more than that every blacksmith develops an occupation neurosis in the arms, or every foot-worker one in the legs, or every speaker one of the larynx, etc. In fact, eye-strain headaches are much overdone, and many so-called eye-strain headaches have really nothing whatever to do with eye-strain, notwithstanding the fact that a careful ocular examination reveals very definite anomalies.

The anomalies which are most frequent are astigmatism, asthenopia, hypermetropia, and emmetropia. The headaches rarely begin until after many years of compensatory overactivity of certain of the eye

muscles, when a real occupation neurosis steps in the overstrained muscle group, showing itself in orbital and frontal headaches, at times associated with spasmodic or other neuralgic phenomena. Sore eyeballs and blepharospasm are among the commonly observed phenomena.

Again, it should not be forgotten that labyrinthine disturbances, even though very slight, are capable of producing the same type of occupation headaches in the eye-muscle group as may arise from anomalies in the eye structures themselves. The function of orientation in space, of which equilibrium is a part, is a compound function in which the eye muscles are closely associated with the cerebellar apparatus of equilibration. This association, anatomists tell us, takes place through the posterior longitudinal bundles and collaterals between the vestibular tract and the fibers from the oculomotor nerves, and is established for the purpose of making use of the eye muscles. Therefore, a continuous series of adjustments take place in the eye muscles, purely automatic in nature, in cases where there is any defect on the vestibular side of the apparatus of equilibrium.

This is frequently overlooked, and many eye anomalies are corrected when in reality more attention should be given to the ear, or possibly to a low-grade labyrinthitis.

Whether we are able at the present moment to separate these equilibration occupation headaches from the pure eye-strain headaches is an open question, but now that attention has been called to them, it seems certain that they will be differentiated, and proper means of adjustment found. The recent work of Barany and others in the functional testing of the labyrinth will help in this diagnostic problem.

Still another group of these occupation neurosis headaches concerns itself with those in which compensatory adjustments take place in the muscles of the neck. These adjustments are necessary, in view of eye anomalies, to bring about a correct picture formation in the retina. The head being held in a compulsive direction, by reason of the eye anomaly, soon brings about a deep-seated occipital, sometimes a parietal headache, with stiffness, which may be relieved by the proper eye measures. Many of these parietal and occipital headaches are found in desk workers, in literary workers, and whereas the primary anomaly may be in the eye the compensatory muscular movements have taken place, not among the eye muscles, but usually in the muscles of the neck.

Quite similar occipital headaches are due to other motor adjustments of the head, due to ear disturbances, possibly to disturbances of smell, and more particularly to bad adjustment in the position of the whole body, which is brought about by the relationship of desks or work benches, or machines to sources of light and to the distribution of the working material. Many women in shops, factories, etc., develop these headaches as the result of too circumscribed and forced motor positions. The adjustment of the various muscular loads has not been considered, nor has the proper elimination been thought out,

with the result that a number of headaches occurring among workers in these occupations are found to be due to these continuous muscular strains.

Not only do we find headaches here, but backaches and other forms of occupation neuroses are frequent. Seamstresses, shop girls, factory hands and others working in positions in which the factor of muscular activity enters show these headaches in profusion.

In another place I have said that these forms of headaches are by no means infrequent (Forcheimer's treatment), but it should not be forgotten that the human body is rarely a symmetrical organ, and to seek for ideal symmetry is more or less of an *ignus fatuus*. Thus, it is idle to argue that all of these headaches are due to eye-strain. To uphold such a doctrine implies a psychical squint, far more serious perhaps than an ocular one, for such a mental squint leads to a perversion of the logical faculties. It develops, when present in mild grades, various types of cranks, both in and out of the profession. In its severe grades one finds the delusional enthusiasts, not all of whom are properly taken care of. From a purely philosophical point of view, it should be remembered that the faddist, the crank, and the delusional enthusiast afford splendid shelter behind whose opinions the charlatan can fatten unmolested.

Treatment.—It is well recognized that the treatment of many of these conditions is often most satisfactory. The proper fitting of glasses may do the trick, but it is often not the only factor, so that a careful study must be made. The history of the patient's occupation must be carefully inquired into, the position taken during the working day, details of the motor adjustment all carefully studied. A complete analysis of the motor habits of the individual must be made, and proper correction made if anomalies are found. It is not enough simply to write a prescription for a pair of glasses when it may be found that if other factors were corrected there would be no need for the glasses.

Myalgic Headache.—This is a type of headache which Edinger has called "indurative" headache. It is a form which has received special attention from the hands of many Swedish investigators. It has been written upon extensively by Cornelius, Peritz, and others, and is a form of headache to which many irregular practitioners have fastened themselves to their advantage, since massage and physical manipulations have given the best results in the therapy.

These results have been obtained, because it will be found that on careful palpation of the muscles of the head, particularly at the tendinous aponeuroses of origin or insertion of the muscles there will be found slight thicknesses or nodular resistances. These are excessively painful to the touch, and continuously painful, although the pain is apt to be diffuse rather than localized. Just what these small nodules or hardnesses are has not been definitely established, although some authors have thought they have shown that they consist of infiltrations of mineral salts. Fig. 14 shows the localization of many of these indurative areas. It may be, indeed, that in many respects they cor-

respond to points described by Valleix one hundred years ago. Valleix thought that they indicated points of special nerve tenderness, and he was comparatively right. It would appear that a new study of Valleix's points would be worth while, separating them from the points of induration, and also from the reflex painful points of visceral disease.

These indurative headaches, or myalgic headaches, are often severe. They may be so agonizing as to resemble meningitis.

FIG. 14



The points upon which "indurations" are most frequently formed. (Edinger.)

Diagnosis.—Oftentimes one gets the impression that one has a brain tumor to deal with, but the absence of eye signs and the distinct relief that follows a deep massage after finding the indurated points, establish a correct diagnosis. The diagnosis of tumor is often made because there is very frequently pronounced nausea from the excessive pain. As a rule there is no elevation of temperature.

Palpation of the forehead shows no hyperesthetic areas in the parietal and temporal regions as is often the case with the migrainous headaches. One finds at the insertions of the muscles at the back of the neck, occipital tender points, and often in the belly of the muscle there are to be found small nodular swellings. These nodular swellings may be,

as it were, rubbed out by deep massage, both from the tendinous insertions and from the belly of the muscle.

Not only does one observe acute attacks of headache as a result of these indurations, but a chronic, persistent headache is even more frequent. These chronic headaches are usually occipital. They show a very persistent intermittent course with acute exacerbations. They will start with an acute attack, then disappear more or less. The patient will have a chronic headache which will more or less pass away, and then there will be an acute exacerbation, and then a more or less chronic occipital distress. The pain, moreover, is apt to radiate into collateral regions. The deltoid muscles may be involved. One very frequently finds that these patients have also attacks of muscle soreness in other parts of the body; that they have lumbago or wry-neck or a stiff shoulder.

This feature is very significant, especially when small, nodular, painful swellings are found in other muscles than those of the back of the neck. It is very significant, furthermore, that exposure to cold is found in these cases as an etiological factor. This gives countenance to the belief that they are more or less rheumatic in origin. This, however, is purely a lay interpretation. "Uric acid" is the favorite formula of explanation with many, and one hears vague talk of these nodular swellings being due to various forms of auto-intoxications. It may be so, but just what the auto-intoxications are, from whence they arise, and how they produce the disturbance remains yet to be seen. Occasionally one finds a quack masseur, regular or otherwise, who speaks of these as "chalky" deposits which they can rub away, and inasmuch as the therapeutics by deep massage is effective and useful, one is inclined to swallow the pathological explanation having experienced the relief.

Treatment.—Treatment then is preëminently by massage. Certain changes, possibly in the nature of a localized edema, have taken place, in these tendons and muscles, and shortly after the acute stage is over, these localized edemas or nodular swellings should be attacked by general systemic remedies and by local massage. The patient should be given an active laxative, and take a hot bath, or preferably steaming or hot packs should be given. A salicylate preparation, 15 gr. of aspirin, or other related salicylate compound, and then the sore muscles should be massaged, usually with the thumb, and the pressure should be directed toward the flow of the lymph channels.

The massage should be at first gentle, for at least two or three days, then it should become more energetic, until it becomes of the quality known as deep massage. A mechanical vibrator is distinctly advantageous in the later periods of the treatment. Such a massage séance should begin with five minutes and later should extend to fifteen to thirty minutes. Treatment should be at least biweekly, and it may require from four to six weeks to complete the therapeutics. General tonic measures are advisable.

Just what is the origin of these tissue edemas, which seem to be

specific for tendons and muscles, is unknown. It is possible that they may be local disturbances of the deep sensibility fibers, which can cause localized edemas within the body structure, just as localized edemas may result on the skin as in herpes and urticaria from definite forms of irritation either of the posterior spinal ganglia or of the gastro-intestinal tract. Most attention has been riveted on the gastro-intestinal tract, but at present no definite relationship has been established.

In looking at the question of exudation from the bloodvessels, and the increased tendency to permeability of the vessel walls, there seems to be reason to think that the composition of the blood plays a very large role. In other words, the tendency for exudates to take place seems in some manner associated with the chemical composition of the mineral constituents of the blood, particularly of the calcium salts.

While it is more or less fashionable at the present time to regard defective calcium metabolism as a fundamental element in a number of pathological processes, it would seem to be fairly well established that the use of calcium salts has a fairly definite action in diminishing the tendency for minute exudations, particularly as they occur in such disturbances as urticaria, in gastro-intestinal disorders, and following the use of a number of toxins. Therefore, it would seem rational to add to our resources, in the treatment of these indurative headaches, the use of calcium salts, particularly the calcium lactates.

These can be given in free doses of from 2 to 3 grams per day.

Osteopathy has fattened upon many of these patients, simply because the average physician has forgotten all about them. This type of headache is rarely described in the text-books, in fact very few of the little things that bother mankind are to be found in the text-books, and it is because of this neglect of the little things that the irregular practitioner gets his opportunity. It should not be forgotten that Valleix and his contemporaries described these headaches many years ago. Their modern resuscitation has come largely through Swedish investigators.

Eye Headaches.—It should not be overlooked that acute or chronic inflammation of the eye structures themselves may be sources of most pronounced headache. Such disorders within the eye structures have nothing whatever to do with eye-strain. Thus severe headaches result from conjunctivitis, from iritis, keratitis, and particularly from glaucoma. The latter is of extreme importance, since lack of recognition of this disorder may lead to permanent impairment of sight, and injudicious treatment often results disastrously. Headache of glaucoma should not be mistaken for neuralgia. The therapeutic indications for these headaches are very definite but cannot be considered at length here as they belong in the department of eye diseases.

Nasal and Frontal Sinus Headaches.—Either through reflex or direct mechanical irritation, the end filaments of the trigeminus may be affected within the nasal and frontal sinuses.

As is well known, acute swelling of the nasal mucous membranes gives rise to a heavy feeling in the head, and when the exudation is

excessive or persistently kept up within these frontal and accessory sinuses chronic headache is a frequent result.

In the acute forms of frontal sinus inflammations, the headache is usually sharp, located in the frontal region, strictly localized between the eyebrows, and not infrequently the skin in that region is tender to touch, and may also be positively painful. This tenderness to pressure may extend over the malleolar prominences, especially when the accessory sinuses are involved in the acute exudative swelling.

Transillumination affords the best method of determining the degree of swelling within the sinuses. The swollen mucous membranes stand out prominently and obliteration or filling of the sinuses is at once apparent when inflammatory reaction is accompanied by pus formation.

This also is frequently diagnostic more particularly by an extension of the pain, its increase, and the not infrequent presence of pulsation. Transillumination shows the blocked sinuses.

Gros has shown that bony conduction is frequently complicated in these sinus disturbances, and his test by means of tuning-forks, alternately placing the foot of the fork to one or the other side of the nasal bones, frequently gives important information as to the sinus involved. The headaches are often extremely severe, very chronic and difficult to solve.

Nasal polypi may be responsible for some reflex headaches. They often offer obstruction to the circulation within the sinuses, and thus bring about frontal headaches.

The rapid advances in methods of investigation of the accessory sinus renders it important that special information be obtained if a frontal sinus headache be suspected.

Treatment.—Therapeutically considered, one is invariably bound down to symptomatic therapy. Small doses of atropine, combined with aconite, with local astringent and emollient sprays are of service in acute catarrhal cases.

Recent pharmacological investigation has seemed to show that exudate and transudate phenomena are largely dependent upon variations in the mineral content of the cells within and without the vessel walls, and that the mineral salt that plays the chief role in regulating the capacity for exudation and transudation seems to be calcium. This seems to indicate that usually the calcium salts would be of service in the treatment of the acute catarrhs and therefore instrumental in bringing about a relief of headaches.

This is not the place to discuss *in extenso* the treatment of catarrh of the nose, and the suggestion is thrown out solely because of its bearing on the question of acute and chronic headaches due to sinus disturbance. Salicylates are of advantage in the influenzal types. In the case of polypi or other obstructions or of purulent sinus disease surgical intervention is imperative.

It should not be overlooked that in superior nasal operations the dangers of ethmoid infection and brain abscess are real.

Ear Disease Headaches.—Headaches due to ear disease are not infrequent. They are for the most part bound up with earaches or with the pain of mastoid disease. Here the parietal region is the site chosen. The pain is usually widespread, often increased by movements of the jaw, the mastoid is frequently painful to pressure, and the general region should be carefully investigated, inasmuch as a headache in this region, more or less persistent for some time, may be the only sign of a beginning temporosphenoidal abscess following a middle ear or labyrinth disease.

Headaches of a very similar nature are encountered in disease of the labyrinth itself. Here, however, one obtains a number of accessory symptoms, such as labyrinthine nystagmus, which is both rotatory and horizontal, vertigo, tendency to staggering gait, and other signs due to disturbances of space orientation. The Barany tests are needed for purposes of diagnosis.

Treatment.—Treatment here is palliative. When the disorder is catarrhal, belladonna, aconite, and calcium may be employed for catarrhal conditions, whereas surgical procedures are necessary if there is pus or excessive tension.

Bone and Periosteal Headaches.—These headaches are not frequent. They occur as complications frequently of caries, syphilis, gummas, and in traumas. They are closely localized. Tenderness to pressure and palpation afford ready means of diagnosis. As a rule the symptoms are not referable to the headache alone. The treatment is purely etiological.

INTRACRANIAL HEADACHES

Having considered the more important of the extracranial headaches, attention should be directed to those of the intracranial region. Here one can easily distinguish four large subdivisions, although it should be borne in mind that the dividing lines between them are by no means sharp. These are:

1. Headaches due to disease of the meninges proper.
2. Headaches due to new growths within the cranial cavity, giving rise to pressure within the ventricles or within the brain substance, acting either upon the cerebral structures or upon the meninges.
3. Those due to disturbances in the blood supply of the brain or to modifications in the amount of cerebrospinal fluid within the cerebrospinal axis.
4. Thalamic headaches.

As can be seen, pressure upon the meninges is a common factor in all of these disturbances, but as has already been pointed out the meninges are not the only structures containing pain receptors, although possibly the only cerebral structures containing pain receptors for ordinary touch. The cerebral structures themselves, particularly the bloodvessels, unquestionably contain deep pressure sensibility fibers, so that the pain of intracranial disturbance is a complex of meningeal as

well as of the deep sensibility reactions. Specific pain reactions from the thalami should not be neglected.

Meningeal Headaches.—Those due to acute disease of the meninges will not be considered here, as they have been discussed already elsewhere. Such headaches are a constant accompaniment of cerebro-spinal meningitis, tuberculous meningitis, pyogenic forms of meningitis, such as follow traumas, middle ear or sinus disease, and the frequent headaches following the postinfectious diseases, such as, for instance, in influenza, typhoid, etc.

These headaches are often very severe. They may occur early in the disease, particularly in the acute pyogenic infections, and may for a time be the only factors present. Thus, for instance, many cases of poliomyelitis complain of severe headache for at least twenty-four hours before the general disease is recognized. Here the process is probably a meningitic one.

Meningeal headaches are usually both frontal and occipital. In tuberculous meningitis it is well known that the pain extends to the neck, and occupies the occiput almost exclusively. The other symptoms, such as rise of temperature, dulness or stupor, stiffness of the neck, various cranial nerve palsies, even slight convulsive phenomena soon establish the fact that these headaches are upon a very definite infectious basis, and do not belong to the category of those with which we are dealing here.

Symptoms.—But there are headaches due to subacute or chronic diseases involving the meninges. These are often severe and occur isolated and independent of other symptoms. It is upon this type of headache that certain emphasis should be laid. These headaches bear the ear-marks of chronic headaches *per se*, and do not for a considerable length of time give much indication of what the causal factor is. Those particularly in mind at the present time are the headaches of pachymeningitis hæmorrhagica interna, pachymeningitis cervicalis hypertrophica, of serous meningitis and of syphilitic meningitis.

It has been recognized for some time that in *hemorrhagic pachymeningitis* headache may be the only symptom. It is usually localized and is persistent. Careful examination of the eye-grounds, however, often reveals the fact that there are swollen disks, or even choked disks, but even this may fail, in which case puncture of the ventricles may be the only means of establishing a diagnosis. In the presence, however, of alcoholism or trauma of beginning senility one may suspect a hemorrhagic pachymeningitis, with the oncoming of severe headache for which no other existing cause can be found.¹

Serous meningitis is often secondary to a purulent meningitis occurring may be many years before. It may be secondary to a frontal or accessory sinus disease, or to a labyrinthitis. Here the headaches are usually persistent. They resemble those of brain tumor or brain abscess, and it is only with extreme difficulty that a diagnosis of serous

¹ Blackburn, Pachymeningitis Externa, Journal of Nervous and Mental Disease, August, 1911.

meningitis may be made. In fact, in the majority of instances the diagnosis is made either surgically or post mortem.

Choked disk is something to be looked for, and stress laid upon the etiological factors already herein indicated.

It is noteworthy that following a lumbar puncture, often following inflammation of the ear, of the mastoid, or of the accessory sinuses, one notes diminution in the swelling in the choked disks. This is by no means an absolute point, but it is occasionally of diagnostic import.

Diagnosis.—The headache of serous meningitis is most frequently mistaken for that of brain tumor. Inasmuch, however, as the indications are operative in either case no serious blunder is made with a diagnosis of brain tumor.

Headaches due to syphilitic meningitis are extremely frequent. They have been studied carefully for years, and show certain features which while not absolutely pathognomonic, yet are sufficiently so to make a diagnosis with comparative readiness. The headaches may come on early or late. As a rule they come on a short time after infection, often much more acute and fulminating in character. They are persistent, violent, either occipital or parietal, and frequently may be delimited by percussion, and are accompanied frequently by skin tenderness. As the tendency of the meningitic process is to spread in all directions, pressure phenomena, such as choked disk, nausea, and vomiting, are apt to be absent, whereas irritative phenomena, as sensory signs, tingling, increased tendon reflexes, fugitive transitory aphasias, cranial-nerve involvements, etc., are much more likely to appear. Thus when one finds widespread, even though fugacious, superficial involvements, with considerable fluctuation and wide symptomatology, one can suspect a superficial, syphilitic meningitis, especially in periods of slight confusion or of mild apathy.

Many of these cases are associated with mild spinal syphilitic spondylitis, and occasionally with symptoms of more serious import on the part of the nervous tissues, namely, Argyll Robertson pupil or other anomalies, irregularities, inequalities, etc.

Examination of the blood usually shows a positive Wassermann, but that of the cerebrospinal fluid is as a rule negative, especially when the usual amounts of serum are used. Phase I, Nonné reaction is variable, as is also the cell count, although slight lymphocytosis, 10 to 50, may be expected.

The headache of a basal syphilitic meningitis presents a certain number of features even more stereotyped than that of the convexity. Here the headache is usually occipital, and it is often violent, and is frequently felt deep within the skull, behind the eyes. In both instances there is a slight tendency for the headache to augment at night, although this old diagnostic criterion of syphilitic headaches is not by any means an absolute one. Tenderness to percussion, strange as it may appear, is very frequently felt most acutely over the eyebrows in a syphilitic meningitis. Here the influence of a contra-coup blow is very striking.

Cranial-nerve involvements are much more frequent in basal syphilitic meningitis. The pupils are often involved, and one frequently finds acute psychotic outbursts, often with a paranoid or maniacal coloring; occasionally the patients are depressed. Polyuria and polydipsia are sufficiently frequent to be striking phenomena. Fever is as a rule absent. Serological changes are those of syphilis of the convexity.

Treatment.—Naturally the treatment is antisyphilitic. Salvarsan is by far the best remedy with which we are acquainted, particularly in those headaches occurring shortly after infection. Inunctions of mercury are advisable, as are also the hypodermic injections of the salicylate of mercury. In the headaches occurring at a comparatively remote date from the time of infection, iodide therapy is also efficacious, and should be combined with mercury or arsenic. Here the doses of iodides should not necessarily run up above 20 or 30 grains a day.

Headache in Tuberculous Meningitis.—In this process headache is an early symptom. As is well known, tuberculous meningitis occurs more particularly in young, poorly nourished children, or in adults who have other tuberculous lesions.

In the former there is frequently a history of antecedent restlessness. Sleeplessness, finicky eating, peevishness, irritability, crossness, and surliness are present. In very young children there is no definite indication of headache, beyond the vague putting of the hand to the head, and the constant fretfulness. Older children complain to the mother of the pain, putting the hand over the whole head. The pain is apt to be intermittent and fleeting in the beginning, but becomes more and more persistent as the disease advances, increasing in severity and in urgency with frequent ups and downs. Emaciation keeps pace with the advancing headache, and there is a constant modification of temperature, consisting of a slight afternoon or evening rise.

Some of these patients develop the more evident signs of meningitis within a week or a month, others, however, develop more slowly, several months in fact elapsing before the outbreak of a definite stupor, delirium, coma, or convulsions.

When in this stage the children lie in a semi-dozed or stupor, throw themselves about, are restless, whining, frequently crying out with a sharp cry, sometimes beating the head and grinding the teeth. An irregular temperature curve is present.

Older patients show less restlessness perhaps, but a dreamy delirium or confusion, with marked indications of cerebral pain.

Tuberculous meningitis *per se* is treated elsewhere in this book; it is only while in the prodromal stages, when the cause of the headache is not so apparent, that it is of interest here.

For the headache *per se* there is no treatment. Diagnosis should be made and the treatment for the tuberculosis be instituted as early as possible. The results, however, are not encouraging.

Intracerebral Headaches.—A large group of headaches due to various forms of chronic encephalitis, chronic ependymitis, to abscess, brain tumor, arteriosclerosis, general paresis, cerebrospinal syphilis, etc.,

may be considered here. In these the pain may be due both to the irritation of the meninges and to pressure phenomena within the cerebral substance itself, with involvement of deep sensibility. This deep sensibility pain is found particularly in arteriosclerotic softenings, in abscess, in the toxemias of alcoholism and lead, and in the persistent headaches of hyperthyroidism so frequent around the menopause period.

The special pains of thalamus involvement need separate consideration. They have characteristics quite different from other intracranial pains, and will be taken up apart. Whether such thalamic central pains may be found limited to the distribution of the trigeminus is questionable, especially to the intrameningeal and sinus distributions of the fifth, but at all events it is theoretically possible that one may get isolated trigeminal central pains due to thalamus lesions, just as it is possible to have pains of the upper extremities, or the lower extremities due to thalamic lesions solely.

Brain Tumor.—The differentiation of various forms of brain tumor which may give rise to headache is not here contemplated. Headache is almost universal in brain tumor, but it should never be overlooked that brain tumor may exist without any headache. Hard, compact tumors are much more often accompanied by persistent headache; whereas, tumors which are of softer consistency, especially those that are infiltrating, such as gliomas, myxomas, etc., may attain considerable size before producing headaches. It is a characteristic of the headaches of brain tumors to be persistent. They rarely intermit, save in the very early stages, and even in the free intervals a certain continuous heaviness is present. They are liable to persist even during sleep. Jars, sudden movement, or conditions which tend to increase intracranial tension almost invariably augment these headaches, and occasionally a therapeutic expedient which will raise arterial tension will provide a clue as to the nature of the headache.

The location of the headache varies considerably, and it is rarely sufficiently well defined to afford diagnostic criteria for localization, although careful percussion may afford valuable signs for this purpose.

It is also observable that a widespread, severe, dull headache may be combined with or associated with one which is more definite and sharply localized, in which case the severer, accompanying pain may afford a valuable localizing sign. To trust, however, to the site of the pain as a certain localizing sign is precarious, for the variations are innumerable. Pontine and cerebellar tumors often give rise to marked frontal headaches; cerebellar growths are known to show occipital and parietal pains; frontal growths have been known to produce exquisite occipital pains.

The definite conclusion is that the site of the pain should always be carefully weighed; it may prove of inestimable value in localization; it may be of no value whatever; it should be, as in all other questions of medical diagnosis, something to be judged in connection with the other symptoms, and not rejected simply because of its insecurity.

As has already been stated, percussion of the head should never be neglected in studying the possibilities of localization of an intracranial growth. Whereas, in perhaps the majority of cases, one obtains no particular information, still there are enough positive cases, which show the sharp, local percussion tenderness closely associated with the involved area, to make this procedure a routine one. Abscesses and cysts frequently show such sharp, localizing tenderness on percussion.

Headache alone should never provide the basis for a diagnosis of new growth, to it, as a rule, the symptoms of pressure in the optic disks should be added. These should consist either of interlacing in the color fields, slight swelling, or the signs of definite choked disk.

Again, however, brain tumor may exist, and yet show neither headache nor optic nerve changes. This is particularly true in infiltrating gliomas, and occasionally true for frontal lobe tumors; temporo-sphenoidal tumors may be present for some time without either headache or choked disk. When, however, the more general symptoms of nausea, vomiting, motor signs, or sensory phenomena are present, then a definite diagnosis of brain tumor can be made and the proper therapy instituted. In the study of the motor phenomena it should be recalled that mild hemiplegic signs, such as increased spasticity, ankle clonus, Babinski, Chadwick, or Oppenheim signs, presence of synergistic power loss, as tested by the Babinski, Grasset, and Hoover methods, should all be employed in arriving at an estimate of involvement of the motor area, either cortical, subcortical, in the midbrain, crus, pons, or medulla.

The sensory signs of intracranial growths are too numerous to be enumerated here.

In all lesions involving the sensory paths, within or below the thalamus, sensory disturbances are more or less definite and clear. Pain, touch, thermal sensibility, localization, bony sensibility are, one or more, apt to be involved, but in lesions above the thalamus the sensory disturbances are usually more difficult of interpretation. They are very much less definite, especially light touch. Astereognosis may be present in involvement of the sensory paths anywhere in their course. Cerebral astereognosis is readily distinguishable from that due to peripheral disturbances.

The exact topographical localization of brain tumors, leading to their surgical removal, is not contemplated in this chapter. This subject has been considered elsewhere in this book. Suffice it to say that treatment, save in the case of gummata, is manifestly surgical. A word may be said, however, about gummata, which may give all of the phenomena of brain tumor, especially when found as single, compact, hard, nodular masses. These may be present in almost any region of the brain, and it should not be forgotten, as has already been intimated, that surgical removal may be a much more expeditious and satisfactory procedure than treatment by mercury, iodides, or any other antisiphilitic remedies. This is particularly true when the gummata are large and when they are in the cortex.

The treatment of cerebral abscesses is manifestly surgical, as well as the treatment of cysts. In abscess and in cysts an antecedent history of infection, trauma, suppurative ear disease, suppurative sinus disease is all important. Abscesses and cysts may lie dormant, even for years.

Hydrocephalus.—An increase in the amount of the cerebrospinal fluid results from a variety of causes. Such an increase may be due to an inflammation of the ependyma, or to purely mechanical blocking of the aqueduct. This latter may come from pressure upon the corpora quadrigemina or pressure on the veins of Galen.

The increase of tension produced by this increase of fluid within the ventricles, independent of its numerous causes, will bring about an intense headache, varying with the amount of fluid within the cerebral ventricles. In congenital hydrocephalus this is not apt to be the case there, for the time being it can be disregarded, and only interests us in this chapter when the process still continues and acute exacerbations occur in the course of a chronic hydrocephalus.

Acquired hydrocephalus headaches are characterized by great intensity and great fluctuations. This irregularity of remission is a fairly constant and striking feature. The headaches are associated with the general symptoms of intracranial tension, choked disks, nausea, occasional vomiting, diminished attention, various degrees of feeble-mindedness or retardation, hebetude, comas, or paralysis. Exophthalmos is often present, nystagmus frequent, and the head may show characteristic signs of bony swelling; the percussion note is often distinctly modified.

Treatment.—The treatment of hydrocephalus is distinctly etiological. If tumors or cysts block the ventricles little can be done, although occasionally the tumor may be approached from the outside. Syphilitic ependymitis, which is a frequent cause of hydrocephalus, should be attacked vigorously. In the more chronic stages the ventricles may be blocked, in which case lumbar puncture or callosal puncture is to be tried. Hydrotherapy, active catharsis, or any measures tending to relieve or limit serous exudation are to be put into operation. Tapping the ventricles and lumbar puncture may be repeated several times, and it has even been suggested that the trocar may be allowed to remain in the cord, or in the ventricle, allowing a few drops to escape from time to time.

HEADACHES SYMPTOMATIC OF TOXEMIAS OR OF GENERAL DISEASE

Toxemias.—Chronic poisoning by lead, alcohol, indican, arsenic, iodine, iodoform, copper, opium, bisulphide, and a number of other substances may give rise to acute or chronic persistent headaches.

The encephalitis of lead as well as those of alcohol, arsenic, and other metallic poisonings are most frequently complicated by a severe nephritis, which in its turn contributes to the headache. Here the

headaches are obvious. The milder cases are described as pressures, heavy feelings, quite analogous to the so-called neurasthenic type of headache. In the more severe cases the headache is extreme, may be associated with mental hebetude, occasionally with convulsive movements. In lead poisoning the color of the gum line, the presence of albumin in the urine, changes in the blood, basophilia, signs of neuritis, all aid in the diagnosis.

Here the therapy is directed toward prevention. Among lead-workers greater cleanliness is the first requisite, for in the vast majority of cases lead salts are transferred from the fingers to the food and from the food to the mouth. Miners working in lead dust or other workers in occupations in which lead salts float in the air should wear special masks.

In those patients in whom the lead gains access through the stomach the use of a very dilute sulphuric acid lemonade is orthodox. It should not be forgotten, however, that many sulphuric acids contain high percentages of lead, and this factor should be eliminated. Hydrotherapy, active elimination, and a diet rich in fat is desirable.

In chronic nicotine poisoning, particularly that resulting from excessive cigarette-smoking, occipital headaches are very frequent. They are due, as all other toxic headaches, to a multiplicity of causes, of which the nicotine is the exciting moment. The changes occurring in the nerve tissues are not well known, but the alterations in blood pressure can be readily demonstrated.

Acute alcoholism and acute morphinism are both associated with severe headaches. In the former hyperesthesia of the scalp is characteristic, in the latter headaches are not infrequently basal, occipital, and associated with much itching, particularly of the nasal mucous membrane and of the skin.

Therapeutic relief is obvious. Emesis and catharsis are indicated. Stomach washing is grateful as well as useful. The headache of an acute drunk should be treated by prompt and continuous catharsis, by eliminative hydrotherapy, by stomach washing, and by feeding. Analgesics, such as antipyrine, phenacetin, or bromides, may be used as surrogates, but are of comparatively secondary importance in the treatment of these conditions.

Nephritic Headaches.—These are caused in part by toxemia, in part by alterations in the circulation within the cerebral vessels. Chronic nephritis with contracted kidney is more particularly associated with chronic headaches. These headaches are in general heavy, dull, diffuse; they make the patients feel miserable. They complain of an inward misery, and such pain in conjunction with the presence of albumin in the urine, and diminished secretion of urea, of high blood pressure, possibly of an albuminuric retinitis establish a diagnosis.

Treatment of nephritis itself does not come within the purview of this work, and it should be sought for in works on general therapy.

Diabetic Headaches.—They are of the heavy, diffuse, pressure type, not infrequently associated with sharp neuralgias throughout

the body; the arms and the face being sites of special predilection. Diabetic coma is usually preceded by increasingly severe headache.

Diagnosis is founded upon the accessory symptoms; the finding of sugar in the urine, high specific gravity of the urine, thirst, itching of the skin are among the most valuable of these accessories.

The treatment is naturally for the underlying condition.

Leukemic Headaches.—Headaches of leukemia are also of this same type. Vertigo, fainting, other signs of anemia, and diarrhea are present. Occasionally a nodular leukemia will give rise to brain-tumor symptoms. Arsenic is almost the only help that we have. Salvarsan is of some value in the more malignant leukemias.

Anemia and Chlorosis.—These conditions give rise to severe headaches, especially in young girls, particularly when poorly nourished and overworked. This is found among factory hands, among industrial workers. Headaches are usually continuous; they involve the entire head, and are either dull or intensely severe, sufficient to interfere with the working capacity of the girl. It should also be borne in mind that it is in this class that the frequent taking of headache powders is especially disastrous, since the majority of these powders contain acetanilide or closely related drugs which have a definite action upon the iron oxygen interaction in the red blood cells. Drugs of the aniline series fix this interchange and thereby diminish the functional capacity of the iron in the red blood cells. Inasmuch as the amount of iron, as well as the number of red blood cells, is diminished the further reduction in the functional capacity of that which is there aids and abets the original difficulty.

One should therefore urge especially against the indiscriminate taking of headache powders in this class of patients. Certain headache powders are not to be despised, especially those containing drugs which do not interfere with the oxygen exchange of the red blood cells.

Therapy, therefore, after diagnosis is established from the color of the patient, color index of the hemoglobin, and the diminished number of red blood cells is a matter of internal medicine, but iron and arsenic play a large part in the therapy. They are not, however, the only factor, because there would seem to be, particularly in chlorosis, a toxic factor, the precise nature of which is not yet appreciated, but which is apparently much relieved by careful gastro-intestinal therapy.

Gastro-intestinal Headaches.—These are for the most part reflex in type. This is best seen, for instance, in the headaches of empty and hungry stomachs. Acute indigestion is frequently accompanied by dull or severe headaches. These are mostly frontal.

In hyperchlorhydria severe headache is not infrequent, and general malaise and heaviness of the head, such as is seen for instance in the mildy seasick, is frequent.

The headaches of constipation are frequent. It is really a sense of pressure that one feels, which is often relieved by free catharsis. Auto-intoxication is not definitely established as yet as an explanation of why; nor are all of these headaches, by any means, reflex; many

of them may be due to pressure anomalies, in which filled or overfilled channels in the viscera bring about disturbed cerebral circulation. Thus the passage of a large stool may give instant relief from a headache. A moment's reflection will show that this could not be due to any relief from a toxin circulating in the blood, but similar reflection points to the relief as an obvious effect upon the circulation and the visceral sympathetic nervous system. The hypothesis of auto-intoxication falls singularly short, especially therapeutically, in the treatment of these headaches. The essential factor is improved motor adjustment of the intestines, and this means careful attention paid to the sympathetic nervous system.

Treatment.—The therapeutics are obvious, but should not consist in the senseless giving of cathartics. Probably there is no more difficult chapter in the whole of medicine than that of the overcoming of certain forms of constipation with their accompanying headaches. Among these, purely psychical constipations, resting upon an anal erotic basis, should not be overlooked, especially as these are among the most chronic of all the constipations, and are less often reached by ordinary modes of therapy. They are fully discussed in the chapter on the Psychoneuroses.

Headaches of hepatitis, cholangitis, and gastroduodenitis are due to infection, toxemia, and fever. They need only be mentioned.

Postinfectious Headaches.—After a number of diseases, particularly tonsillitis and influenza, severe persistent headaches occur. These headaches, particularly postinfluenzal headaches, may be of great severity, and when combined in an individual with overwork, the resulting malaise may be extreme. Such a headache and such malaise often create the impression that the patient is suffering from a tumor of the brain. This impression is very frequently strengthened by the fact that not infrequently a mild neuritis, due also to the influenza, will produce a monoplegic or hemiplegic muscular weakness.

These headaches are usually located in the back of the head. They may be dull or sharp, sometimes resembling the pains of a neuralgia. It is characteristic that effort increases them, especially intellectual effort, such as reading or doing any mental work. The moment that one ceases to carry on the intellectual work it not infrequently occurs that the headache diminishes in intensity, or may become only an underlying dull sense of heaviness within the head. During sleep or walking the headache may not be noticeable.

Such postinfluenzal headaches often persist for weeks, or even a month or more.

Treatment.—The proper treatment is by means of elimination of the toxin as rapidly as possible, which is best carried on with hot baths, active catharsis, massage of the entire body, particularly of the back of the head, and frequent feeding. In some cases the patients cannot rest in bed and a modified isolation treatment is impossible. Here graduated walks, automobiling, carriage riding is useful. Avoidance of all work is a *sine qua non*; the patient is unable to attend to any

business. If the economical situation warrants it, nothing is more advantageous than a sea trip.

Analgesics and bromides may be used to mitigate the severity of the pain, but opium should be avoided under all circumstances.

Syphilitic Headache.—Headache is a frequent symptom in cerebrospinal syphilis. If the process is active the headache is intense, if latent, the headache is apt to be dull but persistent. Other diagnostic signs in this disorder are an advancing neurasthenia, pupillary changes, serological and cytological findings, which may precede for months, or even years, not only the headache, but also the more obvious neurological signs. Not infrequently in the early stages of a syphilis, even during an eruption, headache may be intense. These early headaches are not infrequently hemicranic or migrainous in character. They are also often occipital; they show a tendency, even in the early stages, to intermission and remission, or variations in intensity just as do the later headaches of syphilis, especially increasing toward night. There is a tendency for such headaches to disappear in the daytime.

Greater variability even marks the headaches of later cerebrospinal syphilis, and the nightly exacerbations are not so persistent in the later stages as they are in the early ones.

In general paresis this symptom is extremely variable. Many patients have no headaches at all; many complain of a disagreeable pressure, and as a rule such complaints are made only in the beginning of the disorder. In the later stages of general paresis, headaches are rarely permanent features of the symptom picture, save following convulsive seizures.

Treatment here is naturally to be directed against the syphilis.

PSYCHOGENIC AND PSYCHOTIC HEADACHES

A large group of headaches falls under this general category. Under the former may be grouped all those headaches which certain individuals develop either as an habitual selfish reaction to avoid exertion or as a defence interposed in response to any interference with individual egoistic plans. Such headaches of course are very useful as explanations for refusals to accept the little so-called courtesies of social intercourse, but they have a great disadvantage in that, being frequently indulged in, they have a tendency to recur at intervals, which are not altogether appropriate to the individual's plans. They therefore contribute to not only increase personal selfishness, but they induce a state of diminished resistance to more distinctly physical causes of headache.

One can also include with justification the large group of headaches due to the desire for alcoholic or drug stimulants. The patient having once tasted of the relief obtained by the use of alcohol, morphine, or other narcotics tends to create situations which demand a repetition of the pleasure obtained by the remedy. This form of headache is not infrequent, especially among the well-to-do. In the life of the demi mondaine as well as the haute monde it is almost universal. "Really,

my dear, I must lie down; I have such a headache." This is the usual formula which precedes disrobing, a dose of a drug, a cocktail, or a whisky, an erotic novel, and a lazy, self-indulgent hour or more.

Treatment of this type of headache is extremely difficult. The medical sycophant usually has great success. The more stern and ideally inclined practitioner finds himself out of his element in attempting to handle this class of individual.

The headache excuse-habit is a universal phenomenon. One reads of it in all autobiographies; one hears it being constantly discussed in the street cars; it is the chief theme in most polite conversations, and forms the burden of complaint whenever conversational topics run low. Few people go through life without some headache, but no one has as much as he complains of having. The headache excuse-habit needless to say is pernicious. As a widespread symptom of general dishonesty it is revolting to the physician; as an efficient excuse it is worn out.

One may say that the majority of the so-called neurasthenic headaches, occurring in those individuals who do not know what to do with their time, are really excuse-habit headaches. An individual runs out of energy, begins to be less efficient in his labors, does not want to push himself or drive himself in order to do high-grade work, and then rigs up a headache to explain it. Mankind is constantly excusing itself for its inefficiencies and deficiencies, and the easiest place to lay the blame is in a headache.

Neurasthenia Headaches.—These are real things, but as isolated signs they are extremely rare. Just as a headache with stiff pupils, positive Wassermann, positive globulin and cell count in the cerebrospinal fluid means cerebrospinal syphilis, so a headache, in order to be a neurasthenic, must show accompanying and definite factors, characteristic ergograph tracings, in the muscular sphere, defects in attention, loss of power in addition experiments, and a whole series of neurological reactions which the labors of laboratory workers have established. The diagnosis of neurasthenia should only be made after carrying out such a series of examinations. Every organic factor should be rigidly excluded.

Simon-pure neurasthenic headaches are rare, and rigid psychoanalysis frequently shows that emotional factors play a greater part in their development than does overwork. It will be found, as a rule, that these neurasthenic headaches are mostly the headaches of anxiety neuroses in which the unsatisfied libido, libido being used in a broad sense, occupies a prominent etiological position. Moreover, worry concerning finances, conduct of children, love affairs, unalterable bitterness, economic sordidness, etc., are but a few of the emotional factors that occupy a far more important role in the production of neurasthenic headache than does overwork.

The true neurasthenic should have behind it toxic or fatigue factors. The postinfluenzal headaches, already written upon, could be considered true neurasthenic headaches, while those just enumerated in

which a large emotional factor is present may also be included under this rubric, because of the particularly fatiguing effect of continued emotional stress.

Symptoms.—The prominent feature in true neurasthenic headache is the sense of weight in the head. This is of course not pathognomonic, but it is always to be borne in mind. The pain is rarely acute, the head feels heavy, some patients call it woodeny. The pressure is usually occipital, but may be frontal or situated anywhere in the head. It not infrequently goes from place to place. The number of descriptive phrases used is innumerable. One speaks of iron bands about the head, another says that he is carrying a heavy helmet, another can hardly hold the head up on the spine, etc. In the anxiety neuroses headaches, dizziness, or giddiness is not at all infrequent, the patient feels as though the head were empty—as though things turned around.

Another feature of the true neurasthenic headache is that it is often worse in the morning; it may clear up in the afternoon or evening; is made worse by talking, writing, or by effort requiring concentration. Emotional excitement, if prolonged, raises these headaches to a high pitch.

Irritability is another general sign; the petty pin pricks of life are born less gracefully, and slight exhibitions of temper, often generously called temperament, increase the discomfort.

Hypochondrical depression is not infrequently an accompaniment. It should, however, be separated from the depression of a mild manic-depressive psychosis.

Treatment.—Treatment of these forms of headache has been already discussed in the chapter on the Psychoneuroses, Vol. I.

Hysterical Headaches.—Pure hysterical headaches in the sense of head-pain conversions in individuals possessing an hysterical character are those here referred to.

The headache excuse-habit is not included within this group. True hysterical headaches are not frequent. Hysterical clavus, with boring pain, sharply localized, is one of the most characteristic types, but there need be no regularity about the hysterical headache, since that which determines the location for the conversion rests upon some incidental factor, possibly a blow on the head, a fall on the head, some minor but emotionally accentuated accident involving the head.

The chapter on the Psychoneuroses, and especially that on Hysteria, considers this subject in detail, and will not be further dilated upon here.

Manic-depressive Headache.—In mild attacks of a manic-depressive psychosis, often spoken of as cyclothymia, one has a characteristic picture that should not be overlooked, because in patients suffering from this disorder suicide not infrequently takes place, much to the chagrin of the physician.

Mild depression is an underlying feature in these patients, yet they may nevertheless sedulously guard their mental attitude. They do

not permit the outside world, nor even the physician, to know how depressed they are, because they fear being considered mentally disturbed. They therefore project their mental depression upon their bodily organs. Gastro-intestinal disturbances and pains in the head are the most frequent symptoms complained of. These patients often show other mild physical anomalies; they may complain of their eyesight, or of the throat, or of the nose. Backaches, urinary disturbances are frequent, and they often complain much of constipation. The physical is enhanced throughout, in order to conceal the mental. Hypochondriasis is the older rubric that would envisage many of these manic-depressive headaches.

Careful study, however, shows that these patients are usually slow in their reactions; they talk and move with less promptness than is their usual wont, saying in explanation, if attention is called to the fact, that the headache or the indigestion is responsible for it. Intelligence tests, however, Bourdon, addition, etc., show not the characteristic neurasthenic curves, but those of retardation. Further search into the history will also reveal other "neurasthenic" attacks, perhaps some periods of busy activity and excessive well-being, not infrequently a distinct outburst of excitement, or a frank depression, which the relatives will attempt to explain away as a "depression" over a love affair, or "neurasthenia" over financial worry. The family history may show similar periodic outbursts, of a mild or a severe grade, in collateral branches; mild alcoholic episodes may also point in the same direction. The inference is that these headaches are really signs of a manic-depressive psychosis of a mild grade.

Treatment.—Treatment is, in the first place, protective. Other features are discussed in the chapter on the treatment of the Manic-depressive Psychoses.

Dementia Præcox.—Hypochondriacal headachy ideas are very frequent in beginning dementia præcox in many patients. Frequently they appear under the guise of "neurasthenia," or the modern symbol psychasthenia. Many complain of an empty-headedness. It is one of the characteristic features of the beginning of the habit disorganizations upon which Meyer lays so much emphasis, and rightly, of the shut-in personality described by Hoch, of the useless day dreamings, and half-baked philosophizing of the predementia phase, spoken of by the present writer and others. Certain of these dementia præcox headaches are of immense theoretical importance. It not infrequently happens in this disorder that the patient has an interesting and complex explanation for his headache. It is carefully mapped out. He often can draw a picture of it. Such a description will not infrequently contain very suggestive material of a richly colored symbolical significance. These are significant and often diagnostic.

Treatment.—A complete psychoanalysis gives the only hope of aiding such patients. This should be made in the early stages, as with the frank outbreak of the disease treatment is possibly of little avail. (See Chapter on Dementia Præcox.)

OPHTHALMIC MIGRAINE

Symptoms.—The symptomatology of this affection, also frequently spoken of as hemicrania, sick headache, bilious headache and the like, is too well known to need detailed description here. At the same time it is not as widely recognized as it might be that migraine attacks masquerade under other forms, and for this reason it seems advisable to say a word about the so-called classical migraines, as well as of the abortive and aberrant types.

An attack of classical migraine usually begins with a feeling of chilliness; with eye symptoms, blurring, floating scotomas, which advance to partial blindness. The patient, after fifteen to twenty minutes, commences to have a severe headache on one side of the head, or over the whole head, which is accompanied by throbbing, by nausea, perhaps by vomiting, by inability to work, with hot flushing sensations throughout the body, and a sense of distress. Vomiting, which may continue for hours, usually terminates the attack, and after a period of sleep the patient makes a complete recovery. The attack may last an hour or two, a day, or occasionally two or three days.

Numerous studies have shown that this type of attack is the exception rather than the rule. Patients will have attacks of this kind throughout a lifetime, but they will have other shorter attacks, which will not be recognized as migraine; oftentimes in large numbers, with comparatively infrequent attacks of the severer variety.

In an outline in Osler's *Modern Medicine* I have stated the opinion that migraine is a very widespread disorder—almost universal. In an analysis of nearly three thousand continuous patients at the Vanderbilt Clinic, Department of Nervous Diseases, less than 10 never had any kind of headache, and in all save about 50 the headaches were certainly migrainous in character. Although fully fifteen hundred claimed at first that they never had migraine, yet careful further questioning showed that at frequent intervals, once in three or four or more years, perhaps, nearly all had had true migrainous attacks, while in the intervals abortive and aberrant migrainous headaches were comparatively common.

The older classics on migraine have regarded only the severe attacks, and therefore one finds so many notions regarding heredity and the like.

If the present attitude be correct that migrainous headaches are more or less universal, then one's judgment concerning their frequency should be based on the abortive and aberrant attacks rather than upon the severe ones. Then it can be seen that the general attitude toward migraine must change very materially. Regarding the aberrant and abortive types, comparative studies have shown them to be extremely diverse. There are attacks of migraine which consist of chilly feelings only; there are attacks with chilly feelings and scotomata; there are others with scotomata alone; there are some with scotomata and headache; there are some with headache and chilliness; there are attacks

of vomiting alone, which are purely migrainous; there are attacks of flushings alone which are migrainous; there are even migrainous, aphasic attacks recently described under the head of intermittent claudication; there are some pseudo-epileptiform states, fainting states, etc., which are fundamentally migrainous in their nature, and one might go on expressing the variants which a migrainous patient might reveal.

There have not been enough studies of individual patients published. Analyses of every attack occurring in a migrainous individual are desirable, for they show how rich the variable symptomatology may be.

Etiology.—This is not the place to discuss the etiology, for the various hypotheses have been numerous, nor can it be said that at the present time any one hypothesis will explain all of the phenomena. A comprehensive review of these is to be found in the article referred to. The present drift of opinion would seem to indicate that the vasomotor hypothesis is in general the most tenable. This, as modified, means that for one reason or another, and these reasons are numerous, the sympathetic control of the cerebral bloodvessels is modified, and there results in the classical program a constriction of the cerebral bloodvessels. There follows a modification in the flow of the cerebrospinal fluid within the ventricles, intraventricular tension, possibly cerebral tension increases, causing pressure and the headache, which pressure finally being relieved the attack is over. This general hypothesis of intraventricular tension as well as intracerebral pressure is not as yet subject to physiological proof, but the analogies and homologies with other known facts concerning intracerebral conditions justifies the conclusions.

Treatment.—The problem of treatment then must be viewed in its largest aspect. If the general point of view herein laid down is correct there is no such thing as a treatment of the diathesis. There is no migraine diathesis or constitution. Migraine is a more or less universal attribute or possibility, occurring in severer forms in certain individuals than in others and appearing in severer attacks than others in the same individual at certain times. We therefore believe it to be senseless to treat a patient for a migrainous constitution, and furthermore, believe that an appeal to heredity is a subterfuge which permits one to put on intellectual blinders, which prevents one from seeking for those individual factors in the environment which are responsible for the upset in the intracerebral tensions.

Therefore, no general treatment is rational which is not individual. The laying down of general laws is superfluous if not farcical. There is no question but that in some individuals emotional excitement is the initial impulse, while in others the matter is of an entirely different character. To treat both in the same way, or to regard both in the same light, and to be met with the same class of therapeutic approach invites failure in at least 50 per cent. of the cases. Insistence should be made upon ascertaining all the individual factors provoking attacks.

It is an oft-repeated observation, which now has the sanction of many centuries behind it, that most migrainous patients get well as they

grow older, that is to say, migrainous attacks are not infrequent in youth, becoming less and less frequent in middle age. This may mean simply an habituation of the nervous system to certain forms of stimuli; when naturally they cease to be effective, or possibly the ability of the brain and the brain spaces to handle varying degrees of intracerebral tension gradually improves.

On the other hand it is found that certain migraines begin in later years, and, under the same formula of interpretation, one must assume that new conditions are arising which interfere with the adaptability of the cerebral structures to take up these variations in intracerebral pressure. Pathologically this would seem to be the case with these migraines of later years, for in a large majority of cases it is found that an advancing arteriosclerosis is the most important factor.

Not infrequently other organic disease of the brain is present; and in all these later migraines it is important to bear in mind the possibility of a cerebral tumor, occasionally syphilitic gummata.

This is not the place to take up the etiology of many of these so-called symptomatic migraines, for one can say that, as a rule, late appearing migraines are apt to belong to this so-called symptomatic class. They represent the general inability of the cerebral structures to handle variations in tension, brought about by vascular modifications, but point to more or less specific factors in bringing about such a disharmony.

Not infrequently these migraines are diabetic, others are nephritic, others are present with exophthalmic goitre, particularly of the so-called larval or abortive types. Thus many of the migraines of the menopause are expressions of hyperthyroid activity. Naturally in any scheme of treatment the symptomatic relationship must be sought for and handled. The migraine, as such, is of secondary importance.

Seen then from this broader point of view, the therapy of migraine becomes more hopeful than when viewed from the standpoint of an hereditary affection. While the viewpoint here maintained demands a closer scrutiny for pathological factors it promises greater opportunities for successful therapy. Naturally, then, in the majority of cases, the treatment resolves itself into the control of the attack as such, followed by a search for whatever accompanying factors there may be. Such accompanying factors, as has already been indicated, may consist in those forms of irritation, which acting upon the vascular mechanisms, produce the disturbing changes in intracerebral tension, or, as in the case of the symptomatic migraines, it becomes essential to bring into relief actual disturbing factors in the brain tissues themselves or in other organs of the body apart from this hair-trigger, vascular situation.

Individual experience will in large measure influence the mode of thought regarding the so-called peripheral types of migraine. There are many who, interested in one or other specialty in medicine, have considerable experience in the treatment of migrainous attacks, which seem to be induced by some apparently trivial peripheral disorder.

It is extremely difficult to express any composite view concerning these peripheral excitation factors, for the eye specialist is apt to maintain that all migraines are due to eye-strain; the reflex nasal paths account for all those seen by the rhinologist; while the neurologist is prone to believe that he can find an emotional cause for each and every upset in the vascular equilibrium. The truth probably lies somewhere between these extremes, and there seems no reason for doubting that each and all of these types of peripheral factor play a part in the production of some migraines.

Accumulated experiences through many years seems to point to the gastro-intestinal tract as the most important of these peripheral malefactors, but if one bears in mind how important a function nutrition is for the body, and how, theoretically at least, one-half of man's activities are centred about his gastro-intestinal tract, it is very evident why this portion of the body has appeared so important in the production of so many different disorders. An historical survey of medicine shows that the gastro-intestinal tract has been the great citadel from which have emerged nearly all of the ills to which flesh has been heir. With advancing years this storehouse of woes has been slowly depleted, and now, apart from purely organic disease in the gastro-intestinal tract, one finds that one has to take refuge in vague terms such as auto-intoxication, enteroptosis, sluggishness of intestinal movements, etc., to maintain the supremacy of this old Bluebeard in the etiological domain.

Superstition dies hard, and the amount of magic still intertwined about all medicine is difficult to eliminate.

The practical inference to be drawn from these considerations is that where peripheral disturbances are found they should be corrected. It is absurd for a patient to have to wear glasses, or to have painful operations on the nose, or to have to wear an abdominal belt, or have an intestine looped up here, or a kidney fastened there, to have a uterus tilted to the right or to the left, up or down, to have an ovary excised or what not, just because the patient has an attack of migraine once in three or four years, or even once in three or four months. If the various peripheral mechanisms are sufficiently distorted to bear a definite causal relationship to a migraine, then constantly operative causes should induce more constant effects. A floating kidney, a loose intestine, an unbalanced eye does not get out of gear once in three or four months and produce its migraine. If it has anything whatever to do with migraine the chances are that the migraine will be much more constant, because if a cause at all it will be much more persistently active. The procedures outlined rather briefly therefore have no warrant in the treatment of the migraine alone. When producing other disturbances as well as migraine they may be considered, but such consideration does not belong to this chapter.

Certain migraines unquestionably have been helped, if not entirely cured, by the correction of these peripheral disturbances. They are few, however, and have been for the most part usually mild, or in

some severe cases the peripheral disturbance—eye-strain, ear-strain, constipation—has been extremely marked.

Certain gastro-intestinal factors need to be emphasized, however, even bearing in mind the warning concerning one's psychological attitude toward the stomach. Certain articles of diet are certainly tabooed for many persons, but in these cases the patient usually has a fairly good idea himself of the situation. One should pay proper attention to these dietary restrictions, yet every patient should be tried out on all his beliefs. One should put to the test the ideas that the individual has with reference to certain prescribed or tabooed articles, and see for one's self whether the belief be well-founded or not. In this field one will meet with some extremely interesting experiences, and it will be found that a few migraines seem to bear a very definite correlation to reactions which are closely allied to the so-called urticaria or serum-disease reactions, which in the domain of dermatology are of so much theoretical as well as of practical importance. (See Vol. II, Chapter X, for a discussion of these reactions and vascular disease.)

These migraines bear a very definite relationship to certain types of foods, and the migraine follows with almost mathematical regularity the ingestion of these foods, just as is well known for instance that certain individuals cannot take strawberries without having an urticaria. Just what the food is for the particular individual cannot be stated *a priori*, nor is the connection between the food and the reaction always of the same kind. Thus, certain individuals develop migraine after an excessive carbohydrate intake. This is not, strictly speaking at least, a form of reaction allied to the serum-disease reactions just alluded to, it possibly has relation to excessive fermentation and production of alcohols or other related chemical bodies. Along a similar line of thought one must consider the ingestion of alcohols. Here there is very frequently a relation between alcohol intake and migraine. This is particularly true, as is well known, for champagne, and for the large majority of individuals the dry champagnes produce more headache than the sweet ones, notwithstanding the more or less general belief to the contrary.

One cannot here discuss the various alcoholic drinks, because the reaction is nearly always individual, and one must inquire rather carefully into the experiences of the individual referable to alcoholic intake and migrainous aftermath.

Excessive fat intake—sausages, fried food, and the like, so-called rich food—often bears a definite relationship to migraine and it is not at all unlikely that for certain individuals certain foods should be tabooed.

All of these questions are as yet matters based upon very insecure foundations. Our ideas concerning them are descendants of century-old feelings. We are unable as yet to explain the chemical or biological rationalism, and are therefore guided largely by empiricism. Such empiricism, however, has always been found, when viewed in the large, to contain psychological truth if not actual truth, and until such actual

truth in terms of chemical formula can be demonstrated a certain amount of consideration must be given to the empirical beliefs.

If such factors as malaria, gout, lead poisoning, etc., can be demonstrated to be present naturally these should receive their proper treatment.

It is the author's attitude that there is little reliable evidence to support a belief in what is known as a migrainous constitution, and that therefore complicated rules of living, with senseless restrictions of all kinds, are valueless. They are more productive of semi-invalidism than of good health, and this general line of attack upon a migraine, namely, of overscrupulous, finicky regulation of the entire life history of the individual, is worse than the disease itself. Therefore, no formal diet charts, no time cards for getting up or going to bed, nor rigid formulæ for exercise are advised in this chapter. This does not mean that one patient in ninety-nine may not be benefited by such. It simply advances the argument that the ninety-nine should not go through a senseless regime because we, as physicians, are unable to pick out the one.

Treatment of Attack.—This is on the whole fairly satisfactory. There are few migraines, even severe ones, which cannot be benefited.

Individuality again is essential in the treatment of the attack. Although unequal in action, there are a number of vasodilators which are useful at the very onset. The nitrites and nitrates which have been in use for many years can frequently be employed with success, but a careful choice must be made, while it is essential that the severity of the attack be well studied in considering the dosage as well as the remedy. Slowly acting nitrites, such as the sodium salts, are valueless. Probably a mixture of nitroglycerin and erythroltetranitrate will be found to produce the greatest effect. The powerful character of the nitroglycerin gives immediate relief, while its evanescence is counteracted by the steadier action of the erythrol, the slower working of which maintains the effect. As stated, slowly acting nitrites are of no value. By the time they begin to take effect, cerebral pressure has been reduced by vomiting, etc., and the stage of usefulness of the vasodilating remedies has gone by. Nitrates and nitrites are practically worthless, excepting only in the vasoconstriction stage, and in many individuals, for reasons as yet unknown, they have no remedial effect at all. When administered too late they increase a later vasodilatation, and only cause increased pain.

Analgesic vasodilators are found to be of the greatest service. An exact knowledge of their chemical structure should be gained in order to obtain the best results. Each item of their action must be carefully studied, for one must take into consideration the fact that although they are closely related in their general effects, there are specific differences in the pharmacological action of these analgesics. Precise information on these points is therefore necessary to insure the highest efficacy of treatment. A long and ever-increasing list of these remedies gives ample opportunity for adaptation of the remedy to the individual

patient, always bearing in mind that not only must the dosage be correlated to the severity of the attack, but that tolerance to any particular drug is bred by frequent repetition in use. It is well therefore to vary the analgesics. Antipyrine, acetanilide, phenacetin, pyramidon, and the related salicylic compounds (aspirin, etc.) are all useful, especially antipyrine, which, although not always applicable, is particularly valuable because of its rapidity of solubility and action. Acetanilide, either alone or in combination with related analgesics, is of service, as are also the bromides and caffeine, although the latter is very much overrated, and is useful chiefly in abortive attacks or to disperse the remnants of a severe attack. The bromides and chloral may be used in mild attacks, in which they will be found probably sufficient, the more powerful remedies being reserved for more severe attacks, and the treatment being adapted to the needs of the individual occasion.

It is of the greatest importance to be able to modify the analgesic from time to time and also even more valuable to administer a happy combination of analgesics which for the individual attack is most serviceable. The members of this general group of analgesics act in a dual capacity—some even have triple activities to be reckoned with. Thus aspirin acts by reason of the antipyrine within it as an analgesic and vasodilator; the salicylate radical acts as a weak vasodilator and a gastro-intestinal irritant. Its analgesic properties may be highly desirable, its gastro-intestinal irritating activities highly disadvantageous, and more in certain attacks of migraine than in others. Personal experience with its use has seen, at times, a great increase in the gastric symptoms—therefore I rarely rely upon it exclusively.

If the pain is excessive the more powerful analgesics are advisable. These are acetanilide and pyramidon—but they must be given in small doses. The solubility of the analgesics to be used is of importance to know. A soluble analgesic acts quickly for a short time; as a rule the greater the insolubility the greater the delay, and to a certain extent the more prolonged is the action.

It is of little value thus to use an insoluble analgesic for short migrainous attacks, and of less value to utilize the soluble ones exclusively for the longer attacks.

Combinations, therefore, are advisable. It is impossible to outline the individual variations of the analgesics here. A reliable pharmacology should be on every practitioner's shelf. Such a work as Cushny is invaluable. Two evils should be mentioned with reference to these analgesics—yes three—and the last shall be mentioned first, *i. e.*, drug habit. Many people get so in the habit of utilizing these analgesic drugs that they fly to them on the slightest approach of a pain or a discomfort. Again, certain analgesics by reason of the wide vasodilatation produced cause a disturbance in the cardiac mechanism. They are called cardiac depressants, for this and other reasons. Pyramidon and antipyrine are those that need the most careful watching in this respect. Another drawback is with those containing the aniline

radical, acetanilide being the type. Here there results, from sufficient dosage, a locking of the iron, whereby its reaction with oxygen is interfered with. Cyanosis and poisoning may result from this diminution in the oxidizing capacity of the blood. Continued usage may bring about chronic suboxidations—the importance of which is not yet accurately known.

The key-note then should be small doses in combination and the avoidance of excessive repetition. It is an economically absurd position to take that a physician should be called every time one has an ache or a pain. The taking of analgesic drugs cannot be stopped if this alternative, *i. e.*, calling in the doctor, is the only one; the patient doses himself in order to save expense. Hence in dealing with a migrainous individual—and here in this chapter all are conceived as such—there should be a free discussion of the whole situation with the patient, economic factors, when necessary, being taken into full consideration. The patient should be warned regarding the analgesics. Such a warning should not proceed from the unconscious-advantage-to-be-gained motives, but from a real understanding of each individual's human problem.

The combinations most frequently found advantageous for the individual case should be carefully recorded. Personal experience has found some of these combinations to be as follows:

For quickly passing evanescent abortive attacks:

R—Antipyrini 3j
 Caffeine citrate gr. xv
 Elixir sodii bromidi q. s. 3ij
 3j q fifteen minutes for three doses.

R—Pyramidon, gr. v, with cup of hot coffee.

If the attack is in the chilly, creepy stage, with beginning scotoma and before the headache:

R—Antipyrine gr. xv
 Nitroglycerin gr. $\frac{1}{16}$
 Elixir lactopepsin q. s. 3j
 3j q five minutes for three doses.

R—Aspirin, gr. v q ten minutes with cup of coffee.

If the attack is well on and belongs to the more obdurate types:

R—Antipyrine, 3j
 Acetanilide 3ss gr. v

Capsulæ vel tab., no. x. One tablet, repeat in fifteen minutes. Take cup of coffee, hot footbath, and lie down in darkened room. At end of hour if pain still persists and if no vomiting:

R—Aspirin 3j
 Elixir sodii bromidi q. s. 3ij
 3j, repeat in twenty minutes, two doses.

R—Pyramidon 3j
 Acetanilide 3j
 Caffeine citrate 3ss
 Elixir sodii bromidi q. s. 3ij

3j q twenty minutes for three doses; repeat in two or three hours if not relieved.

In the migraines of later life chloral hydrate in doses of from 2 to 10 grains may be used to advantage. In the menopause migraines, with marked disturbance of vasomotor control, bromide mixtures are of great value, as are also combinations with small doses of aconite and belladonna. Here, as has been mentioned, careful study of the thyroid function should be made. The giving of fairly good doses of the thyroid, as an experimental procedure to determine if hypothyroidism or hyperthyroidism be present or not, is warranted; also a trial of ovarian or spermatie extracts. Such trials should be made under strictly controlled conditions. To tell the patient what they are getting—calling it something new and of great value—perhaps implying one's own originality and genius as having found it out, this is psychotherapy, if not something worse, and vitiates the interpretation of the results. All people are more or less influenced by their physician. The rapport that exists should be utilized for the patient's benefit and not be converted into coin of the realm.

The migraines of arteriosclerosis are treated from the standpoint of the vascular disease. Their consideration is found in the chapter on Senile States in Vol. I, and of Cerebral Hemorrhage, Vol. II, chapter X, where a full discussion will be found. Other organic migraines, syphilitic, brain tumor, ependymitis and the like, have been discussed elsewhere in this and other chapters.

A few other remedies recommended highly in previous days are entitled to consideration.

Although *cannabis indica*, or *cannabis americana*, has a definite, if limited place, this drug as well as aconite, which is now rarely used, has been practically superseded by the newer and more efficient analgesics. Yet the addition of cannabis will often prove of service in attacks with which much mental depression is associated. Care, however, must be taken in the selection of the preparation, as it is an extremely variable product; tablet preparations especially, as a rule, being worthless. This may also be said of the volatile nitrite preparations.

In no case should opium or morphine be used except as a last resort. As a rule there is no occasion for its use; the analgesics have robbed migraine practically of its terrors, and it is rarely needed.

In the severe exhausting attacks a brisk saline laxative should be administered, and the patient should be put to bed in a quiet darkened room and well covered, the avoidance of cold being important. A very hot bath will often be found an aid to comfort.

Above all, each patient must be treated individually.

CHAPTER V

THE TREATMENT OF SPASMODIC DISORDERS

By HERMAN H. HOPPE, M.D.

THE group of disorders considered in this chapter have little intrinsically in common, though on the surface the resemblances are often marked. Conditions varying all the way from the so-called functional to well-marked organic diseases are included. The only warrant for including such diverse disorders for treatment in a separate group is a purely practical one. Various disorders, some well-defined diseases, others hardly more than clinical symptom-complexes have this in common—that they are predominantly motor affections, and further, that in many instances they look sufficiently alike on the surface to make it necessary to keep this whole heterogeneous group in mind in order to make a correct diagnosis—a necessary pre-condition to a rational therapy.

TICS

In order to approach the subject of the treatment of these various affections, it is essential to draw a sharp line of differentiation between spasms and tics.

A spasm is a reflex act, manifesting itself in a clonic convulsion of a single muscle or a group of muscles. It is the product of a change, an irritation in the muscles or nerves of the spinal or bulbar reflex arc. Tic on the other hand is a psychoneurosis. It is a voluntary contraction which has become a habit. "The synergic and coördinated muscular contractions of tic imply a cortical origin," says Brissaud. "Among the many varieties of spasm, clonus, hyperkinesis, it is impossible not to recognize certain motor affections, certain movements of defence, of expression, of mimicry, certain gestures more or less coördinated, for some imaginary end, all readily distinguished from spasms, fibrillary contractions and choreiform or athetotic movements. It is only logical to attribute a somewhat more complex origin to these varying gestures, in which the influence of the will, however unperceived in the end, is always to be detected in the beginning. The will may not play a conscious role, but the cortex alone is capable of imitating such acts."

That tic, for instance, is of cortical origin can be illustrated by the habit of blinking, and we might contrast it at the same time with facial spasm. In facial spasm we see a unilateral tonic contraction of all the muscles supplied by the facial nerve. In tic blinking we see a

bilateral contraction of the orbicularis palpebrarum, as if in response to a corneal or retinal irritation, a movement of defence. If the contraction of the orbicularis palpebrarum is a lower motor neurone reflex, why is the spasm limited only to a part of the muscles supplied by the facial nerve? Why are not the others involved? If we presume that only the nuclear cells or the fibers going to the orbicularis palpebrarum are involved, why is the contraction bilateral? The movement makes the impression that it is premeditated, purposeful, and the bilateral movements are coördinated, and hence they are of cortical and not of reflex origin. Tic is always associated with a mental process; there is always a psychological aspect to the affection, and it may be associated with other evidences, or may be the only indication of an abnormal psychic state.

Definition and Characteristics.—Brissaud says that tic may be defined as a physiological act, functional and purposeful in character, which has become a habit, purposeless and meaningless in its execution. The movements in tic are limited not to the muscles supplied by a single nerve trunk, but to muscles physiologically grouped for a special function. There are two separable elements in the constitution of a tic: (a) A mental defect; (b) a motor defect.

(a) A certain degree of mental instability is a distinguishing feature of tic patients. They are not insane, but are neuropaths or psychopaths. The fundamental defect is a lack of inhibition, a weakness of the will. In addition there are often conditions of psychasthenia in the form of morbid impulses and obsessions.

(b) The motor defect consists of the production of a motor reaction by some external stimulus or an idea. At first this external stimulus or idea is necessary for the production of the motor reaction, but by dint of repetition and in the absence of the external stimulus or the idea, the contractions continue habitually and automatically without purpose or meaning. In spite of the fact that the movements are frequent, irregular as to interval, exaggerated and prolonged in character, evidences of coördination and purpose can usually be detected. Tics are preceded by a desire for execution and are succeeded by a feeling of satisfaction. An attempt to suppress tics causes a feeling of distress and dissatisfaction. These contractions occur in attacks, repeat themselves at irregular intervals, they vary in frequency, duration, and degree. They can be restrained by the will or by distracting the attention and usually cease during sleep. Fatigue and emotions usually increase the frequency and severity of the attacks.

A tic is a physiological act, which has become pathological by being evoked in the absence of an adequate external stimulus or a corresponding adequate psychic process. The act is the product of an irrestrainable psychomotor impulse. The will may be able to inhibit the act to a certain degree, but its ability is limited and the attempt is accompanied by a feeling of distress and mental pain, which is relieved by the motor explosion.

Tic may be limited to a single coördinate movement, localized tic,

or we may have a number of contractions occurring simultaneously, or in succession, or we may have a generalized movement.

Etiology.—The underlying basis of all cases of tic is an hereditary or acquired neuropathic or psychopathic condition. The former is the more frequent. It is usually found that tic patients belong to families other members of which are also affected with some form of functional nervous disorder. Moreover, the patients themselves suffer from other manifestations, usually psychasthenic in character, either obsessions or morbid impulses. Fundamentally the nervous system lacks automatic inhibition. These patients are not insane, although tics are common in the insane, and some tic patients later in life develop insanity. Tic usually develops in young children, but may develop at any age, although the onset usually occurs before maturity. Emotional disturbances, bad training in childhood, trauma can all act as exciting causes.

The special form of tic develops usually as a result of an external irritation, or on the basis of some idea or both combined. Thus as a result of a defect in accommodation or of a conjunctival irritation, the child blinks his eyelids; later on the habit is established, the cause is removed, but the blinking continues without reason or purpose. As a result of a tight neck-band, or an ill-fitting coat, the neck is stretched, the shoulder is elevated. This attracts attention, and the patient coughs to divert attention from these movements; soon the neck is twisted, the shoulder shrugged, and the cough repeated—even in the absence of an observer, and after the removal of the irritating cause, and the tic is established.

The evolution of a tic is gradual, its onset is insidious. Meige and Feindel say that it always has a tendency to invade, "being a functional act, it moves in the direction of greater complexity." Muscular groups, accustomed to act in physiological unison are affected together. Tics constantly vary in amplitude, degree, and frequency. "We have our good days and our bad quarters of an hour."

The tic may be limited to one group of muscles, or the same individual develops various forms of tic, which can exist simultaneously, or which from time to time may replace each other. Intercurrent diseases may arrest them, convalescence reestablish them. "A sense of well-being is a panacea (Janet) for the tiqueur." By way of imitation tic may appear epidemically, viz.: The jumpers of Maine, the Latah in Malay, the Myriachit of Siberia.

Varieties of Tic.—Since tics are physiological movements, with purpose, turned into habitual movements, purposeless and meaningless, we may have as many tics as there are physiologically grouped muscular movements. There are some, however, which are more common than others, and these affect most frequently the muscles of the face, neck, and upper extremities, since these muscles are used most for physiological expression of cortical activities, and so we may have facial tics, unilateral or bilateral; tics of the eyelids, of the tongue, of the muscles about the nose, of the muscles of mastication, biting of the nails and

lips, tics of the neck muscles (mental torticollis), tics of the extremities of the trunk muscles, of spitting and swallowing, tics of speech, respiration, etc.

Tics of Speech.—Tics of speech are commonly associated with other facial and neck tics and they are caused by the movement of the air through a more or less contracted glottis associated with spasmodic contractions of the diaphragm. In the absence of organic causes, tics of speech occur when the faculty of speech is not in operation, and usually consist in not only useless but inopportune utterances. Aside from fundamental sounds and simple words, the patient may precede or follow a spoken sentence by words or phrases which are inappropriate, which are ejaculated without intention or control, at all times and places, with no relation to the idea conveyed in the sentence. Other forms of tic are seen in the form of the ejaculation of obscene words or curses (coprolalia) or in the form of repeating words or phrases just heard or spoken by themselves (echolalia). In order that the ejaculation of curses or obscene words may be classified as tics, and not as a mere habit or as a manifestation of some mental syndrome, they must have the characteristics of abruptness, impetuosity, irresistible impulse, independent of time, place, and association. The patient is conscious of the impropriety of the act, but is incapable of resisting the impulse.

Stammering and defects of phonation during speech are not tics. But tic often takes the form of stammering and is usually associated then with other forms of tic.

Maladie de Tic Convulsif.—*Maladie de tic convulsif* (Gilles de la Tourette) is a syndrome which is developed in individuals on a basis of neurotic degeneracy. The tic is slow in its onset, following perhaps a mental shock, limited at first to the orbicularis palpebrarum or some one of the other facial muscles, then extending to the neck and shoulders, and finally involving the trunk and extremities. The tic reaction may occur simultaneously in all parts, or the various tics may succeed each other. Speech and respiration are also affected. Mentally, there occurs obsessions and abnormal impulses, delusions are developed, progressive mental weakness becomes apparent, and finally dementia sets in.

Pathology.—Tic is a psychoneurosis, and the pathology is unknown as it is of other functional nervous states. Meigs and Feindel are inclined to believe that the phenomena of tic represent some congenital anomaly, some arrest or defect in the development of cortical association paths or subcortical anastomoses, minute teratological malformations that our medical knowledge is still unhappily powerless to appreciate.

Diagnosis.—Tic must not be confused with Jacksonian epilepsy. The actual observation of one attack will suffice to rule out the latter condition.

The great majority of cases of tic are very mild in character, the spasms not very violent or frequent and at first sight might be

mistaken for chorea. If we bear in mind that the movements in tic affect muscles physiologically associated, simulating acts purposeful in character, systematized, with more or less long periods of rest between the contractions, we can easily rule out chorea. It is necessary to make the differential diagnosis, because the prognosis and treatment are entirely different in the two affections.

Ticquers are not insane, as the condition may resemble hysteria but only superficially. Tics never disappear suddenly and cannot be influenced by suggestion but only by a prolonged course of habit reëducation.

Prognosis.—The prognosis of tic varies with the age of the individual, the mental capacity, and the methods of treatment. Meige and Feindel hold that the individual can be cured if he can will to be cured. A temporary remission must not be mistaken as a cure, for the great tendency of the ticquer is to relapse or to change the form of the tic movement. Children frequently under the influence of training and correction, at times under the influence of threats, drop these habits. If there is marked mental infantilism or marked mental inferiority the prognosis is bad even under most improved methods. The disease, however, is usually chronic, runs a course of months, and often persists during the entire life of the individual.

Oppenheim reports a case of generalized tic in a young girl which stopped at the first menstruation.

Treatment.—The old idea that tic is incurable is no longer held: many cases are relieved by general and special treatment. Tic itself does not lead to insanity, but in some cases the underlying mental defect may be progressive and lead to dementia.

Surgical Treatment.—We cannot warn too strenuously against the surgical treatment of tics. This warning is especially uttered against the surgical treatment of mental torticollis. In all of these cases the result will be paresis, atrophy, and permanent deformity, without relief to the tic. The contraction will affect other muscles. In one case of the writer's in which not only the spinal accessory but later three or four upper cervical nerves were severed on the affected side, the patient's condition was not only not relieved but spasms continued in other muscles, and the discomfort was increased by the weakened neck muscles. Any successes which have been obtained have been in cases of spasm and not of tic, and this holds for torticollis as well as facial spasms.

Hygienic Measures.—The first indication for treatment in all cases should be a general hygienic one. Hygienic, dietic measures, the regularity of a simple life because these patient are unmethodical and changeable; tonic baths of short duration, simple non-exciting foods and a mental and physical occupation with plenty of sleep are the first indications for treatment. The general health of the patient should be brought up to par.

Of the medicinal agents I have found the regular administration of sedatives at bedtime to be of value in diminishing the severity of the

paroxysms. Bromide of strontium, in doses to suit the age, usually about 30 grains at bedtime, is perhaps the best. Any one of the other sedatives can be used. In the violent cases chloral or even chloroform may have to be used to give temporary relief. However, in one case of severe tic of arm and leg, the use of chloral in sufficient quantities to produce stupor was continued for six weeks without avail. Arsenic is useless. Hydrotherapy is of benefit in the form of hot packs. In children, isolation in the form of a strict rest-cure will often effect a cure. Oppenheim lays great stress upon gymnastic exercises; these are given by competent instructors and are followed by periods of rest. Electricity is of no avail, except as a psychotherapeutic measure. Hypnotism has been reported as a success in several instances, but is usually without avail, except in cases complicated with hysteria. Oppenheim calls attention to the fact that tic patients are usually hard to hypnotize. Non-hypnotic suggestion has only a temporary effect.

Attempts on the part of the patient at perfect muscular relaxation, attempts at keeping the body and more especially the muscle groups subject to tic at perfect rest, attempts to control the automatic and emotional reflexes, should be carried out systematically. The key to a successful therapy is the treatment of the underlying psychasthenia.

Reëducation.—Methods in reëducation have been evolved by various men. These methods are based upon the recognition of the relation between the convulsion and the mental state of the patient. Oppenheim calls his method inhibition gymnastics; Brissaud, Meige, and Feindel, psychomotor-reëducation. Pitres has devised breathing gymnastics. Oppenheim's method consists in training the patient to inhibit reflex, defence, and emotional reflex movements. Thus, he takes a pointed instrument and slowly approaches the cornea while admonishing the patient not to blink. Or the mucous membrane of the nose is tickled, and the patient admonished to suppress the reflex. Again, physical pain or tickling is resorted to with the command not to draw away the part which is prodded or not to yield to the impulse to laugh or respond in any way to tickling. These exercises are all intended to bring into play the will, and to strengthen the power of inhibition and the suppression of reflex movements. The method involves regularity of routine and painstaking perseverance.

The method of Brissaud, amplified and extended by Meige and Feindel is called the method of reëducation. The method consists in a combination of immobilization of movements with movements of immobilization. The patient is taught to preserve immobility he is taught to replace an incorrect movement by a normal one. The exercises must be graduated, short in duration at first. The patient is required to remain absolutely motionless as if sitting for a photograph, first for a few seconds, then for a longer period up to a half hour. The patient must be encouraged that he can and must remain immobile. First place the patient in a sitting position; when this has been done satisfactorily, then in a standing position. When he

can remain immobile for a period of time in this position, vary the position and attitude of head, arms, and legs. Eventually he will learn to maintain immobility of certain parts of his body while he is walking or while he is executing given movements with his arms. All the time direction must be specially paid to the patient's tic.

The method and its object in training inhibition must be explained to the patient, and his interest and intelligent coöperation must be gained and maintained. At the same time there must be instituted the discipline of movements; the movements must be slow, regular, and accurate, and addressed to the muscles involved in the tic. The period of exercise should be short and fatigue must be avoided. These exercises of immobilization and of movement should never occupy more than one-half hour and should be repeated three or four times per day. More accuracy and sustained interest are attained if the exercises are executed before a mirror. In order to prevent relapses the exercises should be continued for a long time after the tic has disappeared. Just as neglect and indulgence on the part of the surroundings fail to correct bad habits and allow them to be evolved into tics, so also indifference and carelessness on the part of parents will militate against the success of the treatment. In some cases antagonistic movements or gestures can be employed. These of course must vary with the tic movements. If the mouth is drawn to the right the patient can be directed to draw it to the left. If the eyelids are batted the patient can be directed to keep them open for an undue length of time. This method of antagonism can be applied to all tics.

Hartenburg advises the use of the faradic current to produce the antagonistic movements (Fraenkel), the method of overcoming passive resistance to the opposing muscles. In all cases care and supervision are necessary to prevent the antagonistic movement itself from becoming a tic. Each tic must be studied and treated by special measures which the physician must elaborate and order. Meige and Feindel, for instance, give the following as an illustration for the treatment of facial tic: Every day, and three times a day, at the same hours—nine, one, and six—the patient is to look at himself for two minutes in a mirror, preserving absolute immobility the while; to read aloud for two minutes, to speak in front of the glass for two minutes, to walk backward and forward in front of the mirror for two minutes. During the ten minutes of these exercises he will endeavor to keep his facial musculature under control. If the tic assert itself in the course of one of the exercises he will recommence the latter, if necessary twice; the third time he will leave it until the next séance. The treatment of stammering for instance by respiratory movement is very successful. A deep quick inspiration followed by a long, slow expiration. Demos-thenes used present-day methods for the cure of his speech defect by declaiming at the sea-shore with a pebble in his mouth.

Pitres' method of deep inspiration for the cure of tic is as follows: "Supported against a wall, with shoulders braced back, the patient is instructed to take slow and deep inspirations, raising his arms the

while, and letting them fall during expiration. This performance is repeated three times a day for ten minutes at a time. A modification of this method is to have the patient recite aloud for a part of the time. The object is the same as in the other methods, viz.: The fixing of the attention on the allotted task, and the suppression of the tic by the fixing of the attention on some other task. The success of the exercises in the individual case depend upon the ability of the patient to concentrate his attention, and the degree to which he can do it.

Games and sports which demand attention, skill, accuracy, and decision should be prescribed. Manual training is of great value, and can readily be made of service on account of the fact, that departments for manual training are a part of most public school systems today. Psychotherapy is the successful carrying out of the plans which have been laid down in the above paragraph. It means that a definite plan of treatment and of movement has been formulated in the mind of the physician, and that the necessity and, above all, the reasons for the plan have been conveyed to and impressed upon the mind of the patient; that the latter has been convinced of the utility of the treatment, and has been instilled with the enthusiasm necessary to develop patience and perseverance without which success cannot be obtained. It consists in influencing the mind day after day, week after week, for months, and even years, in such a manner that the exercises will be executed each day with the same care and precision as they were in the beginning. That successes can be obtained is testified to by many, that only partial successes will be frequent, relapses many, and failures often, is a foregone conclusion when we consider that tic develops in defective subjects.

SPASMS

Definition.—In contradistinction to tic, it is well, from a therapeutic standpoint, to define a spasm, as a reflex muscular contraction, caused by an irritation somewhere within the reflex arc, either in the centre or in the afferent or efferent nerve trunks. Spasms are characterized:

1. By the extreme abruptness of the contraction, as if produced by an electric current.
2. Spasms lack purpose or coördination. They are confined to some nerve area, anatomically limited.
3. They are not modified by the will, attention, distraction, etc.
4. The spasms may be attended with pain. It is not preceded by an irresistible impulse, nor is its consummation followed by a sense of satisfaction.
5. The mental state in spasm is not changed.
6. If the condition persists during sleep it is likely to be a spasm, although all spasms do not persist during sleep.

Spasm of the facial muscles (Remak) constitutes 44 per cent. of all localized spasms. It is a contraction of the muscles in whole or in part supplied by the facial nerve, reflex in character, independent of cortical

or psychic influences, and caused by a dynamic or material lesion of the reflex arc, consisting of the trigeminal nerve, the facial nucleus, and the facial nerve.

Etiology.—In a large number of cases there is a neuropathic condition of the general nervous system present. Owing to the increased irritability of the nervous system as a whole, reflex response to an external irritant is more easily elicited than would otherwise be the case. The source of the irritation is usually to be found somewhere along the distribution of the fifth nerve. Any irritation of the fifth nerve is capable of exciting reflexly a spasm of the facial muscles. Thus, typical spasms are usually seen during a paroxysm of tic douloureux. Painful conditions of the conjunctiva and cornea, carious teeth, diseased conditions of the nose, and the cranial sinuses, irritation anywhere, even intracranially, brain tumor, orbital plate (Oppenheim), may be the exciting cause of facial spasms. Even intra-ocular lesions may produce facial spasms, as the writer has recently seen in a case in which the only cause, aside from general nervousness, was a detachment of the lower part of the retina.

Pressure or irritation of the trunk of the facial in a few instances has been looked upon as a cause of the spasm. In all these cases the question of a simultaneous sensory irritation can be raised. Thus, pressure at the base of the brain by tumor or aneurysm pressing upon the facial trunk, irritation of the facial nerve in middle ear trouble, have been known to cause the spasm.

Any spasm of cortical origin caused by a material lesion is not to be looked upon as a spasm in the sense of this treatise, but as a Jacksonian epilepsy.

We can hardly reconcile our present views of facial spasm with observations in which the exciting cause has been attributed to lesions of the pelvic organs, or to pregnancy, unless we admit the simultaneous presence of a material lesion in the facial arc which of itself was insufficient to cause a reflex spasm, the pelvic lesion or the pregnancy acting in the nature of a secondary cause. In the vast majority of cases, however, no demonstrable irritative lesion can be found, and it is assumed in these cases that there are molecular changes present in the facial or perhaps even in the cortical centre, which render them unstable and incapable of withstanding ordinary, one might say, physiological stimuli. Sometimes direct trauma of the facial nerve trunk causes an attack of facial spasm. Facial spasms are known to follow head injuries. In the absence of a lesion of the cortex, these cases are perhaps a local manifestation of traumatic neurosis and not real facial spasm.

Symptoms.—Facial spasm is usually limited to one side. It may involve all the muscles supplied by the facial or only a few. The digastric and stylohyoid are usually not involved. When the spasm is widespread the platysma myoides may be involved. The group especially often involved is the orbicularis palpebrarum. Sometimes the spasm is limited to a single muscle or to two muscles, viz., the zygomaticus,

the levator alæ nasi et labii superioris, or the chin muscles. The contraction of the facial muscles after Bell's palsy is usually a contracture and not a spasm. The spasm is usually clonic. It is at times very weak, at others very powerful. The contractions are quick, lightning-like, occurring in quick succession, in paroxysms, followed by an interval of rest, in which there are either no contractions at all, or only weak and occasional ones. Often clonic contractions become tonic and are sustained for a short time, so as to close the eye, furrow the forehead, and draw up the side of the face.

The attacks usually occur independent of any external cause, but are usually increased by emotional excitement. Attacks can be brought on by chewing, talking, or by cold air striking the side of the face. Contrary to tic convulsiv they are not influenced by psychic conditions and cannot be controlled by suggestion.

Facial spasms of cortical origin are differentiated from the ordinary spasms, by the fact that the spasms are apt to spread and not remain localized to the facial muscles. In the second place a cortical facial spasm is usually followed by more or less marked paresis.

Stroking or tapping the side of the face is apt to bring on a spasm. The spasm does not interfere with the voluntary or emotional use of the facial muscles, and does not produce any subjective disturbance in the patient.

The course of facial spasms is essentially a chronic one, often continuous, at times, with periods of remission.

Prognosis.—The prognosis varies with the ability to find the underlying cause. If the latter can be found and removed the spasm may cease. In some cases the spasms have been reported to cease spontaneously. The cases which were cured after a paralysis of the facial nerve offer one of the indications for treatment.

Treatment.—The first indication for treatment is to find the cause if possible. The most promising field is the distribution of the trigeminal nerve. Look for gross lesions first. The eyes should be investigated, especially for errors of accommodation and lack of muscular balance. A number of cures have been reported after correction of these errors not only on the side involved, but even on the opposite side.

Next in order come the various sinuses, the frontal and maxillary. Just as a purulent inflammation at times causes a tic douloureux, so it may also cause a facial spasm. The nose and throat may be the seat of the irritating lesions, enlarged turbinated bones, or ulceration. Enlarged tonsils and adenoids should be removed if found. Caries of the jaw bones calls for surgical treatment, ulcerated teeth should be looked for and, if found, should be removed. At times an x-ray investigation of the jaw may reveal broken-off and ulcerated roots.

The ear should also be investigated as a possible source of trouble. In the absence of any gross lesion we may suspect a neuritis or compression of the various branches of the fifth nerve. Thus Starr advises the method of cocainizing each branch of the fifth nerve in turn. In one case he cocainized the supra- and infra-orbital branches without success,

then the inframaxillary branch was cocainized and the spasms stopped. This branch was then removed and the patient was relieved. He reports success in two other cases investigated along the same lines. If pressure on any of the points of the fifth nerve elicits a spasm, this branch may be removed. Mere section of the nerve trunk does not offer relief.

In the majority of cases no obvious irritating cause will be found. All general and medicinal treatment of these cases is without avail. In some cases the use of cocaine solutions in the nose has been found to be beneficial. A determined stand must be taken against the use of this drug, except perhaps in the eyes or ears, where occasional successes have been recorded. But the dropping of solutions in the nose will surely lead to the contraction of the cocaine habit, not only because it affords only temporary relief in a chronic affection, but because many of these individuals are neuropaths and therefore more likely to contract the habit.

Diaphoretic applications, counterirritants, mild galvanism, with the anode on the trunk of the facial and the cathode on the nape of the neck, may afford some relief.

Nerve blocking has been resorted to in some cases with success. This consists in cutting down on the facial nerve and stretching it to a point sufficient to produce facial paralysis. The success of this method is only temporary, although in one case the spasm did not return after two years.

A more approved and equally successful method was devised by Schlosser and frequently resorted to since. Lately, Patrick has reported three successful cases by this method. It consists in the injection of a 70° to 80° per cent. solution of alcohol into the sheath of the facial nerve. The method consists in introducing a protected needle along the stylomastoid process until it reaches the facial nerve and carefully injecting the solution, a few drops at a time, until a facial paralysis is produced. The spasm stops at once, but is replaced by paralysis, and when the paralysis disappears in six or eight weeks the spasm does not return. In the majority of recorded instances the spasm, however, returned in from three to seven months.

Attempts have been made to resect the facial nerve and unite it with the proximal end of the spinal accessory, but the success of this method is in doubt because it has not been attempted very often.

TORTICOLLIS

Spasms affecting the muscle of the neck are common, and notoriously difficult of treatment. Much light, however, has been thrown on this subject by Brissaud, Meige, Feindel, etc., by calling attention to the psychic origin of many if not most of these cases. There is almost a unanimity of opinion today, that if we exclude the cases of torticollis of mental origin, but few cases of real spasm remain.

It is a mistake to look upon the disturbance as "a spinal accessory spasm." The spasm affects the muscles usually of one side, but often muscles on both sides are affected simultaneously. It may be limited to a single muscle, or to a group of muscles either on one side or simultaneously on both sides. The spasms may not limit themselves to muscles supplied by a single nerve trunk, but may involve several nerve-trunk distributions at the same time, or if one nerve trunk, viz., the accessorius, is resected the spasm affects other muscle groups after the operation.

Etiology.—A true spasmodic torticollis in contradistinction to the mental form is caused by some material lesion in any one of the reflex arcs, involving the spinal accessory nerve or the upper cervical nerves. These material lesions may be adenoids, diseased tonsils, or what is perhaps more frequent, an enlarged and perhaps painful condition of the cervical lymphatic glands. This same relationship may exist between middle ear disease and torticollis, or we may have some lesion of the muscles, bones, or articulations of the neck.

Thus muscular rheumatism affecting the neck muscle may give rise to a tonic spasmodic condition (*caput obstipum rheumat.*). Sometimes wry-neck is congenital, due to an injury of the muscles or ligaments of the vertebræ during birth. In this form the muscles become shortened and later on the vertebræ deformed.

Trauma of the neck muscles and ligaments occur occasionally as a cause of torticollis. In caries of the vertebral column the resulting contracture of the neck muscles is not a true torticollis, but it is one of the causes which must always be looked for.

Organic brain diseases, tumors, and cysticerci of the cerebellum and posterior cranial fossa may so compress and irritate the nerves at the base of the brain as to cause spasmodic contraction of the neck muscles.

Ziehen has called attention to what he terms disequilibrating causes of torticollis. As a result of a diseased condition, say of the eye and ear on one side and consequent overtaxing of this special sense apparatus, the equilibrium of symmetrical cortical excitation is disturbed, and the symmetrical innervation of the neck muscles, for instance, becomes unsymmetrical. Thus, in marked errors of accommodation, or in cases of paresis of the external eye muscles there occurs a secondary turning of the head, which in turn produces a permanent torticollis. Intoxication of a general character as well as general exhaustion, say after childbirth, may play an etiological role. This may explain in part those cases which disappear after the removal of diseased tonsils, cases which occur on the basis of alcoholism and metallic poisons, or after malaria, influenza or the acute infectious diseases. The most acute case seen recently by the writer occurred a month after delivery and disappeared after tonic hygienic treatment in a month. We can picture these causes as operating by producing an increased irritability of the neurone centres.

In the vast majority of cases torticollis is developed without any

irritating lesion to be found anywhere along the course of the reflex sensory-motor arc. It develops usually on a basis of neuropathic or psychopathic degeneracy. With this factor as the real cause, emotional excitement, shock, trauma, overexertion of the neck muscles, may be exciting causes of torticollis. In young children, *spasmus nutans* or muscular spasm of the neck may belong to the latter category, or perhaps may be due to reflex irritations arising from the gums as a result of dentition.

Pathology.—In wry-neck we may have pathological changes in the muscle, as, for instance, a myositis, a tear or injury, and subsequent cicatricial degeneration or shortening of the muscles. If the ligaments are the seat of the trouble, they may be torn, or the joints, the seat of trauma. Caries of the vertebral column is usually tubercular. In the majority of cases the pathology is that of tics in general.

Symptomatology.—The spasms occur either tonically or clonically. Clonic spasms, however, may have short periods of tonic contraction with deviation of the head in a fixed position for a longer or shorter period.

The tonic spasms produce typical wry-neck, and keep the head in a fixed position; the attitude varying with the muscles involved. These tonic spasms are seen in rheumatic affections, in traumatic myositis as seen especially in the newborn, and in caries of the vertebræ or injuries to the ligaments and joints of the cervical vertebræ.

The variations in the attitude of the face and head vary with the muscle or muscles affected, and the intensity and duration of the spasm.

The nodding spasms of children are due to an involvement of the rectus capitis and longus colli. Very mild affections of this kind are seen at times in adults, occurring as a simple nod or with a slight rotation to one side, produced by an involvement simultaneously of the deep neck muscles and the sternocleidomastoid of one side. Nodding spasm may be associated in children, with nystagmus, which becomes manifest when the head is fixed. Blepharospasm and strabismus may be associated with it, which would give the whole picture the aspect of a tic.

The muscles which are the seat of the spasm may be normal in size, but in most of the violent cases there is developed a marked hypertrophy. The spasmodic contractions in the ordinary torticollis are not painful, they are subject to exacerbations and remissions, both as to the number of contractions and as to their severity. They may occur continuously or intervals of rest of varying duration may occur.

Differential Diagnosis.—We have touched upon the diagnostic points which distinguish rheumatic torticollis, congenital wry-neck, and the reflex contractures seen in caries of the cervical portion of the vertebral column. As a rule, a tonic contraction or contracture of the muscles with a fixed position of the head, is indicative of a local material lesion.

The most important indication is the recognition of the mental form of torticollis. In many cases this may offer some difficulty. The

absence of any evidence of local disease in muscle, bone, or ligaments, and the presence of an hereditary or acquired neuropathic or psychasthenic condition, together with a determination of the influence of mental and emotional states on the severity and frequency of the spasms will speak for the diagnosis of mental torticollis. This is especially true when the spasm can be controlled by some simple device as putting the finger on the chin or forehead, supporting the face on the palm of the hand, or as occurred in one of the writer's cases, a severe and violent retrocollis could be arrested by lying down, or resting the back of the head against a chair. It is in these cases of mental torticollis that we have a preceding history of mental trouble, or intercurrent states of hallucinatory insanity.

Prognosis.—The prognosis varies with the cause. If the local cause can be found and removed and the habit has not been firmly established, the spasm will disappear. If the spasm is developed acutely on the basis of an intoxication, or general exhaustion, the outlook is good.

The nodding spasms of children have a favorable prognosis. So also the mental torticollis of children. In the adult the prognosis follows the general prognosis of tics. The prognosis in general is bad, but a few cases recover spontaneously. The affection is essentially a chronic one, and may persist during life, subject to exacerbations and remissions, both in the number and severity of the attacks. The recognition of the mental element and the treatment according to the lines laid down under the heading of tics offers the only favorable element in the prognosis.

Treatment.—The first indication in the treatment of torticollis is to find some material cause and remove it if possible.

In the rheumatic form the first indication is rest for the painful muscle. Internally free purgation and the use of sodium salicylate, gr. x-xv every two or three hours, or salipyrine, gr. x, salophen, gr. x, and later on colchicum combined with iodide of potassium are indicated. Local applications of heat in the form of a hot water bag or the ironing method with a small hot flat iron over a moist piece of flannel goods, affords relief. The general health in the ordinary form of torticollis should be brought to par by the use of iron, quinine, and arsenic.

The patient should be kept quiet and removed from all influences which excite, in seclusion, if necessary, to rule out the mental influence of outside observation.

The spasms can be influenced by the use of chloral and bromides taken at bedtime. Morphine and opium are contraindicated in mental torticollis. Their influence is only temporary and the danger of forming habits is great. Curare has been found beneficial, but like opium and morphine should be used only in the violent cases, perhaps during the especially violent paroxysms in cases that are not of the tic variety. Valerianate of zinc, tr. gelsemium, tr. conin have all been used with success. The use of counterirritants on the nape of the neck and back of the ear have occasionally been found successful.

Orthopedic apparatus may be of use in the clonic forms of wry-neck,

but obviously does not reach the cause. Papier-maché collars have been devised, elastic bands so arranged as to exert constant pressure upon the chin and side of face have been used, but usually without much effect. Fixed dressings and apparatus should be avoided except for those cases which are caused by caries or injury to the cervical vertebræ.

Electrical treatment is often used with success. The galvanic current is preferable. If any points of tenderness are found the anode should be placed over them, otherwise the anode is placed over the region of the entrance of the spinal accessory into the sternocleidomastoid, and the cathode over the nape of the neck. Patients usually weary of the electrical treatment, which should be continued for months and even a year. In some cases the use of the faradic current has been followed by success, but it is questionable whether electricity in any form has more than a suggestive effect. If the opponent muscles on the opposite side of the neck are weak from disuse and these increase the deviation of the head, improvement can be obtained by their electrical treatment and massage.

In all cases the treatment described for tic, the method of inhibition of Oppenheim, the reëducation method of Brissaud, Meige, and Feindel, and the respiration method of Pitres should be instituted, together with general gymnastic exercises and healthy out-door sports. Good results have been reported from all these methods. Associated with these methods should be psychotherapy. The patient should be told constantly that he can overcome the habit if he only wills to overcome it and has the patience and endurance to develop this will power.

Hypnotism is usually followed by failure.

Surgical Treatment.—The surgical treatment of torticollis is divided into two methods: (a) The cutting of the tendons of the muscles involved in the spasm; (b) resection of the spinal accessory and the upper cervical nerves.

The method of cutting the tendons of the muscles was devised by Strohmeier, and lately advocated and extensively practised by Kocher. It is indicated in congenital wry-neck, and is apt to be followed by success, if the deformity of the cervical vertebræ is not too great. In most cases, however, improvement can be obtained and freedom of motion of the head secured. After the cutting of the tendons, a plaster-of-Paris collar is to be applied for several weeks, followed by carefully adjusted gymnastic exercises, especially applied to the neck muscles. Kocher does not limit this operation to forms of permanent wry-neck, but to severe forms of spasmodic torticollis. He divides all the muscles affected by the spasm, usually in several operations. In some cases he has cut the tendons of nearly all the muscles, viz., the sternocleidomastoid, cucullaris, splenius, complexus, and inferior oblique. A plaster collar is applied for several weeks and this is followed by gymnastic exercises. Complete success is obtained only in a few cases, partial in others, and failures in about one-half the cases. Success from this operation in the clonic variety is not to be hoped for, because it does not remove the underlying cause.

Resection of the spinal accessory nerve can only be indicated in those cases in which the spasm is limited to the sternocleidomastoid and trapezius muscles, and only then when we are reasonably sure that we are dealing with a true spasm and not a tic. In more extensive muscular involvement it will be necessary to divide the upper three cervical nerves close to the spinal column. Mere division or stretching of these nerves is of no value. They must be resected. Only in a few cases of spasm has success been obtained. The central irritation remains and the spasm is transferred to other muscle groups. The paralysis and atrophy of the muscles lead to a permanent deformity, which, however, may be preferable to the constant spasm. Successes, both in the sense of cure and improvement, have been recorded, but the operation should be considered only in the most severe cases, and after all other methods have failed. Surgical treatment should not be instituted at all in mental torticollis, because it will not only fail, but will leave the patient in a worse state mentally and physically. The cortical condition of the patient will remain unchanged, and the tic will be transferred to muscles on the same, or on the opposite side of the neck.

TRISMUS

A real spasm of the muscles supplied by the fifth nerve in the sense of a local affection of the masseter and temporal muscles is a rare condition. We may have both the clonic and the tonic form.

The result is a firm closure of the jaws with a hard, contracted state of the masseter and temporal muscles. Trismus is usually a symptom of tetanus, tetany, meningitis, or of some organic bulbar lesion.

Functional Trismus.—As a functional trouble it usually occurs by way of autosuggestion in hysteria and neurasthenia, and in this form is not uncommon. Trismus also occurs in the form of a tic, according to Meige and Feindel.

It also occurs in generalized tetany. As a reflex spasm trismus occurs in diseases of the mouth, and is especially seen when there is little or no room for the wisdom teeth, and the latter by their growth and effort to break through the gums are a source of constant irritation. Ulcerated teeth, periostitis of the jaw bones, and irritating ulcers of the mucous membrane of the posterior part of the mouth may cause trismus.

Prognosis.—The prognosis of functional reflex trismus is good, the spasm disappears with the removal of the cause.

Treatment.—The treatment resolves itself into the finding of some irritating lesion and removing it. This lesion will usually be found in the mouth. If there is an irritating ulcer, apply solid nitrate of silver.

An anesthetic may be necessary to explore the mouth for the removal of buried wisdom teeth or the extraction of carious molars. Whatever local cause may be found it should receive its appropriate treatment. In a case associated with an abscess of the masseter muscle the trismus

disappeared when the latter was thoroughly opened and free drainage established.

Usually liquid foods can be taken, if not, feeding through the nose must be resorted to.

Symptomatic treatment resolves itself into the use of bromides and chloral, but is usually not necessary. If the cause is an inflammation of the maxillary joint, local applications of heat and the internal administration of salicylate of sodium or salipyrene is the best treatment.

The hysterical and neurasthenic forms invariably yield to suggestion.

Spasmodic Forms.—Glossospasmus, pharyngismus, spasms in the muscles of the tongue and pharynx are rarely if ever seen in the sense of true spasms. This is especially true of pharyngeal spasms.

Tongue spasms are usually seen in neuropathic individuals, and more especially in hysteria. They may be clonic or tonic, unilateral or bilateral. In the tonic variety the tongue is hard and pressed against the teeth. In the clonic variety it is alternately protruded between the teeth and withdrawn, or it may be moved from side to side in the mouth or twisted from side to side along the long axis. These movements of the tongue, occurring on a neurasthenic or hysterical basis often assume the character of tics. Painful spasms of the tongue and floor of the mouth occur unilaterally in neurasthenic individuals, without any apparent cause, except fatigue. The duration of these spasms is but a half to one minute, and they may not recur for weeks or months.

Rare forms of so-called idiopathic spasms of the tongue have been described. Some of these have the characteristics of localized epilepsy, the attacks being preceded by paresthesia of the tongue. In some of these cases the facial nerve was in part involved, and disappearing after the use of iodide of potassium, left the impression of having been caused by a cortical lesion, probably syphilitic in character.

Hemispasmus glossolabialis is a distinctly hysterical manifestation and not a true spasm.

Purely reflex glossospasmus has been described: once as a result of the presence of a foreign body in the occipital nerve, and as due to caries of the teeth or disease of the pharynx.

Prognosis.—Prognosis is usually good, even though the spasm occurs at intervals of months and years.

Treatment.—If any source of irritation can be found it should be removed.

If the spasm occurs in conjunction with that of the orbicularis oris and is followed by a weakness of the tongue and face, as in Remak's case, a cortical origin should be looked for. Remak's cases yielded to the use of iodide of potassium in eight days. In the majority of instances the underlying hysteria or neurasthenia calls for appropriate treatment. Tic of the tongue as an isolated condition rarely occurs, but should be treated according to the methods of Oppenheim, Brissaud, Meige and Feindel, and Pitres.

Surgical treatment has been resorted to in violent cases, and success in Lange's case followed a unilateral resection of the hypoglossal nerve.

Spasms of the Muscles of the Trunk and Extremities.—There is hardly a muscle or group of muscles of the trunk or extremities which has not been reported as having been the seat of isolated and idiopathic spasms. Wernicke has applied the name of spasm neurosis to cases which he has described, in which many of the muscles of the body were subject to spasms. The muscles of the feet and more especially those of the calves of the legs are especially predisposed to localized spasms.

The majority of individuals suffering from spasms of these groups of muscles are neurotic, neurasthenic, hysterical, or psychasthenic. Intoxication and disturbances of the biochemical mechanisms of the body are often at play. Chronic alcoholic individuals are especially prone to calf spasms during the night. They also occur in lead poisoning, in diabetic cases, as well as during and after exhausting diarrheas. Sometimes the only ascertainable cause is exhaustion after prolonged walking. In at least one case of tonic spasmodic contracture of the peroneus group, the writer was able to give permanent relief by having the pes planus attended to. Reflex spasms may arise from inflamed joints.

Sometimes trauma plays a role. After a fall on the elbow, the writer saw tonic spasms occurring at frequent intervals in the group of muscles supplied by the ulnar nerve. Indirectly, the wound may inclose a sensory nerve and the resulting cicatricial contraction and irritation of the nerve trunk may be the cause of reflex spasms. In the same way amputation neuralgias, due to cicatricial pressure on sensory nerves, may cause localized spasms in the neighboring muscles.

Prognosis.—Varies with the cause, and with the ability to treat the underlying neurasthenic or hysterical condition successfully. Even though the spasms may recur frequently and continue for a long time they eventually disappear.

Treatment.—Wherever a local material exciting cause, an injured nerve, or nerve compressed by a cicatrix, can be found it should be treated according to its special indication.

The underlying hysteria or neurasthenia must be given special attention. Local treatment in the form of anodal galvanism, hot fomentations and massage should be used. Most spasms of the calf muscles yield to a causal therapy, removal of alcohol, treatment of the diabetes, and other causal agents. Bromides and chloral given at bedtime usually afford temporary relief until the cause can be removed. General hygienic measures, gymnastic exercises, general and special for the group of muscles involved, are of undoubted benefit.

Spasms of the Respiratory Muscles.—We have seen that spasms of the diaphragm occur in chorea, in tetany, and tic. They may be clonic or tonic. Occurring as reflex spasms they are very rare. Mediastinal tumors, other diseases of the mediastinum, and pericardium

irritating the phrenic nerve have been known to cause spasm of the diaphragm. Brain affections, meningitis, may also cause these spasms.

Both the tonic and clonic forms are seen usually on the basis of hysteria, and may then continue for a considerable period of time, even weeks and months. Tonic spasms of the diaphragm have been described. The type of respiration then becomes thoracic. Respiration is quick and shallow, the patient suffers from great dyspnea, the abdominal movements of respiration cease, the diaphragm is low, and the epigastrium is prominent. This condition in a mild form occurs in hysteria and in the severer forms in tetany and tetanus.

Treatment.—Ordinary attacks of singultus or hiccoughing can be arrested, as a rule, by arresting all action of the diaphragm for thirty or forty seconds. This can usually be accomplished by taking a deep inspiration, and then slowly counting up to forty or fifty, during expiration. The same result can be accomplished by slowly sipping and swallowing small amounts of water as long as possible without breathing.

In the hysterical form of hiccoughing it may be necessary to remove the patient from home surroundings. At times a threat to do so is efficacious. Counterirritants in the form of burning with the Paquelin cautery, the application of blisters, use of strong faradic currents over the epigastrium are all efficacious and usually act by way of suggestion. Sedatives may be necessary in the form of chloral and bromides; cold douches to the epigastrium may act reflexly by exciting full deep inspirations. Rhythmic tractions of the tongue have been found successful. The application of both the faradic and the galvanic current (anode) to the phrenic nerve in the neck, with the indifferent electrode over the epigastric region, is often successful. The most important role is played by psychotherapy; by the constant assurance that the spasms are harmless, that there is no organic lesion, and that the spasms will cease. Judicious neglect, removal of solicitous friends and relations, and a constant effort to occupy the attention of the patient will in all cases lead to a cure.

Oppenheim has reported success in some cases by the use of bismuth, which, of course, presupposes that an irritability of the gastric mucosa can be an exciting cause in some cases.

When singultus is a symptom of a grave organic brain disease, or of chronic Bright's disease, it does not call for symptomatic treatment. The same is true when it is symptomatic of mediastinal, pericardial, or hepatic diseases.

TETANY

Known in the literature by various names, tetanilla, idiopathic muscle spasms, intermittent tetanus, etc., the disease described below has been known as tetany since Frankl-Hochwart's classical and exhaustive treatise in 1887, although the name had been previously used by Corvisart in 1852.

Definition.—Tetany is a disease whose symptom-complex consists essentially in intermittent, bilateral, tonic muscular contractions, which are usually painful in character and affect with predilection the muscles of the hand and forearm, usually without loss of consciousness.

Etiology.—While tetany may assume a number of different forms our knowledge is crystallizing itself into two facts: (1) That the underlying cause in all forms is a hypofunction of the parathyroids which is the predisposing cause; (2) toxins, either developed in the body or introduced from without, which act as the exciting causes.

The first cause, hypofunction of the parathyroids, means a derangement in internal secretion, and resulting defects in metabolism. In the second, the toxins may be biochemical in origin, viz.: The post-operative tetanies, or the toxins may originate from many sources and produce the clinical neuromuscular symptom-complex on account of the deficiency or absence of the specific secretion of the parathyroids, which would normally neutralize them, or they are introduced in such quantities as to overwhelm the capacity of these glands.

Etiologically considered Frankl-Hochwart's classification will give us a survey as to causation. He divides the causes into (1) acute; (2) chronic recurring—then into tetany: (a) children; (b) adults.

TETANY IN CHILDREN.—The tetany in children is usually associated with malnutrition. Healthy children are rarely if ever affected. It is usually associated with gastro-intestinal disturbances of a chronic character, and with rachitis. This form of tetany is seen everywhere. It usually affects infants, although older children may be affected, one of the writer's cases being ten years of age.

TETANY IN ADULTS.—In adults we have:

1. *Idiopathic Tetany.*—This has been described above. A distinct infectious cause for the epidemics or endemics which occur from time to time has not been found. Whether a soil poor in limestone in one locality, during the period of the year when the ground is frozen, leads to epidemics, which are never seen in localities rich in limestone formation, is merely a suggestion thrown out by the writer.

2. *Tetany of Gastric and Intestinal Affections.*—This form is usually seen in gastric dilatation, due either to atony or pyloric constriction. It may occur in gastropnoia, and then the cause is perhaps a mechanical closing of the pylorus. It occurs also in chronic gastro-intestinal disorders. These cases have been held by some to be reflex. Gerhardt and others could elicit attacks by percussion of the distended stomach; others have reported attacks brought on by vomiting and by the introduction of the stomach-tube. The most frequent cause is perhaps the altered metabolism produced by a deficient digestion and absorption of food, plus the generation, by fermentation, of some toxin in the intestinal canal. The last two factors play an important role as exciting causes in the tetany of infants.

3. *Tetany of Acute Infectious Diseases.*—This form is usually seen at the time of the year and in localities where tetany is endemic and epidemic, and usually affects with predilection shoemakers and tailors.

Here the specific toxin of the infectious disease, typhoid, tonsillitis, cholera, la grippe, pneumonia, etc., is the exciting cause.

4. *Tetany of Pregnancy, Parturition, and Lactation.*—This form is rare. We know that during pregnancy and lactation the internal secretion of the ovaries is in abeyance, and that there is usually an increased function of the hypophysis, and at times of the thyroid. There is a greatly increased metabolism of the body as a whole, and the occurrence of tetany probably indicates a disturbance of the balance of metabolism, due to a want of proper function of the parathyroids.

5. *Tetany Due to Drugs.*—Tetany caused by ergotin, chloroform, morphine, alcohol, phosphorus, and lead perhaps merely indicate that these chemicals act only in the presence of hypoparathyroidism. Pineles was able to cause a recurrence of tetany in individuals who had recovered from an attack by the subcutaneous injection of tuberculin.

6. *Tetany after the Removal of the Parathyroids.*—When tetany was first observed after the removal of the thyroid glands it was supposed that the removal of the latter was the cause of the tetanic attack. We know, however, today that the lack of the specific secretion of the thyroid produces an entirely different clinical picture, viz., cachexia strumipriva.

There is an overwhelming consensus of opinion today that the underlying factor of tetany is an insufficiency, or an absence of the function of the parathyroid glands. The most recent work in this field has been done by MacCallum and Voegtlin, who have shown that a deficient action of the parathyroids causes a deficient calcium metabolism of the body, and that the hyperexcitability of the neuromuscular apparatus is due to a deficiency of the calcium in the blood.

There is still some opposition to the theory that parathyroid insufficiency and defective calcium metabolism explains fully the genesis of tetany in all its forms. Oppenheim agrees with Jacobis, that various toxins, originating without, or developed within the organism, can produce the disease.

7. *Tetany Complicating Nervous Disease.*—The tetany, complicating other nervous diseases, like Basedow's, syringomyelia, brain tumors, etc., is infrequent, with probably the same underlying factors of parathyroid insufficiency plus an autointoxication.

Pathogenesis.—One may say that tetany is a functional disease of the nervous system, produced by an organic change in the parathyroids. The fundamental identity of all forms of tetany was advocated by Chvostek, and this view is almost universally held. The predisposing condition is hypoparathyroidism, which may be congenital or acquired. As a result of this minus function, there is a defective calcium metabolism and inability of the system to bind toxins which in turn produce a state of hyperexcitability of the nervous system. In the absence of the toxin the tetany may be latent.

Symptomatology.—The clinical manifestations are chiefly motor in character, but there are also general symptoms, sensory, mental, and trophic phenomena. The underlying condition is a state of hyperexcitability of sensory and motor neurones.

Muscular Cramps.—These cramps are almost always bilateral. They are tonic in character, intermittent, lasting from a few minutes to several hours, and are usually painful. There may be only a few spasms in all, or the spasms may occur frequently during the day, with periods of intermission, varying from a few minutes to hours. In the majority of cases these spasms are localized to the small muscles of the hands and forearm. The thumb is adducted, at times across the palm; the proximal phalanges are flexed, the distal extended, giving rise to the obstetrical hand. In more severe cases the hand is flexed, the arm bent upon the forearm, and at times the whole arm adducted and crossed in front of the chest. In about one-half the cases (Frankl-Hochwart 70 to 122) the lower extremities are likewise affected. The foot muscles are involved in such a manner as to flex the toes on the sole of the foot and bring the foot into extension (talipes equinovarus). The leg and thigh muscles may also be affected. These cramps occur spontaneously or as a result of mechanical irritation. Walking may bring on the spasms in leg and thigh. In a few cases the entire musculature may be affected, including the masseters, neck, trunk, and respiratory muscles, including the diaphragm. In children, spasm of the glottis is often associated with the other muscular cramps. According to some writers, laryngospasm represents in all cases a mild form of tetany, viz., latent tetany (Kassowitz). Oppenheim thinks that this view is exaggerated, that the doctrines of the intimate relation between tetany, rachitis, and laryngospasm in childhood needs further elucidation, but holds that this relation does exist in many cases.

The spasms are at times easily overcome, at others, offer great resistance, and the attempt to overcome them is very painful. These spasms come on at times without premonitory symptoms, at other times there is tingling, paresthesia, and formication in the affected region. There may be general malaise, headache, and vertigo. The cramp comes on gradually and slowly relaxes; paresthesia and a certain amount of muscular weakness may continue for a little while, or may be present during the intervals (Oppenheim). The temperature is usually normal except in the postoperative cases, when it may go up to 101° or 103°. The pulse is usually increased in frequency during the attacks; the mental state is usually normal, although unconsciousness may occur in cases combined with general convulsions. In atypical cases there may be strabismus and pupillary contraction, with loss of light reaction, due to the involvement of the eye muscles. Lately, Falta and Kahn have described tetany of the gastro-intestinal system.

Theoretically increased irritability of the motor neurones should extend also to the sympathetic system, and Falta and Kahn have shown by actual experiment that the spasms of tetany are not confined to the voluntary muscles, but that the condition of abnormal

irritability extends to the sympathetic and autonomic nervous systems. They have been able to demonstrate a tetania viscerum which manifests itself by increased irritability of the internal organs and by actual symptoms of irritation. Falta was able to show, for instance, that a given dose of pilocarpine would have but little effect on a normal stomach, but that in tetany the same dose caused acute dilatation and cessation of the peristaltic action as proved by the x-ray. They have also demonstrated by x-ray pictures that spasms of the stomach with a patulous pylorus can occur in tetany. They also suggest that tetany of the viscera may exist as the only manifestations of tetany, and refer these to an abnormal function of the parathyroids. They conclude that important modifications of metabolism occur, as a result of the effect of tetany on the vegetative system.

The increased irritability of the motor and sensory nerves manifest themselves through the following phenomena: (a) Trousseau's sign, the production of an attack by pressure on the brachial artery or plexus; (b) Chvostek's sign, due to an increase in the mechanical irritability of the motor nerves; (c) Erb's phenomenon, due to increase in the electrical irritability of the motor nerves; (d) Hoffman's sign, an increase in the mechanical and electrical reactions of the sensory nerves; (e) Schlessinger's sign, spasm produced by stretching nerves (sciatic, brachial plexus).

Postoperative Tetany is in a class by itself. Since the relationship between the parathyroids and tetany has been experimentally demonstrated, improved technique in the surgery of the thyroid glands has greatly reduced this complication. Postoperative tetany comes usually in from two to five days after the operation. In some obscure cases (Dock) after operations in the vicinity of the parathyroids it may occur after the lapse of months. The spasms in the hands are followed by generalized spasms, often convulsions, respiration becomes rapid and labored, there is increased salivation, albuminuria at times, stupor, delirium in some cases, coma, and death. There is some elevation of temperature. The pulse is not much accelerated.

If the removal is not complete or the injury not severe we have what Halsted terms subtetanic hypoparathyrosis, which resembles the ordinary tetany. Some cases occur in which but slight and few tetanic spasms are observed and which yield to a single or a few doses of calcium lactate. In these cases we can assume that the changes in the parathyroids were slight, perhaps due to pressure or bruising during the operation, or to passing disturbances of circulation.

The tetany of pregnancy is usually mild. It may occur only during labor, when the head is on the perineum, or it may occur during lactation.

Sensory Phenomena.—These are usually subjective; objective disturbances are not found. Pain is present during the spasm in many cases and is always caused if an attempt is made to overcome the contracture. Paresthesias are the rule, either preceding the spasm or

during and after, radiating in the affected region. They are present in the form of tingling, formication, or numbness. In so-called latent tetany these paresthesias may be the only symptom, or they may precede the actual onset of the spasms.

Reflexes.—In some cases they have been found wanting, but if changed at all they are usually increased. Mydriasis and paralytic pupils have occasionally been noted.

Temperature is usually normal. Many cases show a slight elevation, especially in children, but this may be accounted for by the complications on the part of the intestinal tract or lungs. Respiratory changes result from the involvement of the diaphragm and auxiliary muscles of respiration. Death at times occurs with symptoms of inspiratory dyspnea.

Trophic Phenomena.—Cataract has been described as one of the trophic phenomena. Increased salivation is seen in the traumatic cases. Hyperidrosis, polyuria, albuminuria, glycosuria may all be an expression of the fundamental disturbance of internal secretion, and metabolic changes. The same may be said for erythema, localized edematous swelling of the skin, joint effusions, urticaria, herpes zoster, falling out of nails and hair, skin pigmentation, all of which have been occasionally described.

Psychic Disturbances.—In the ordinary cases there is no marked mental disturbance, although increased excitability is seen in the chronic cases. In postoperative cases there may be delirium, stupor, and coma.

The most constant findings are hemorrhages in and hemorrhagic degeneration of the parathyroid glands.

Course, Duration, and Prognosis.—This of course must vary with the particular form and exciting cause of tetany. Trousseau divided non-operative tetany into three groups: the mild, the severe, and the grave. In the ordinary endemic or epidemic variety (Frankl-Hochwart) the duration is usually from four to fourteen days, (hospital cases); often the attack lasts two or three weeks, in a few cases two to four months.

In the mild cases the spasms are confined to the hands. They are of short duration, recur a few times in the twenty-four hours, may run along in this way for weeks and months, and probably recur again in the following year. The attack may last only a few days.

In the more severe attacks the cramps are more extensive, longer in duration, attended with more pain, and probably involving the glottis and respiratory muscles, causing severe dyspnea. In the grave cases the intervals between the spasms are of shorter duration, and the patient may die, or the trouble become chronic in character.

The course of tetany in gastric dilatation is usually toward a fatal termination, unless there is surgical intervention, or relief of the dilatation.

The postoperative tetany has been considered above.

The prognosis as to life varies with the form of tetany, and the

ability to relieve the underlying condition. Frankl-Hochwart does **not** consider the prognosis good in children. This is self-evident **when** we consider the underlying diseases in these cases. Ten children **were** affected with tetany in the Cincinnati Hospital, and of these **three** died, one with widespread pathological changes depending upon congenital syphilis, one of bronchopneumonia, and the third of an inspiratory dyspnea which was not relieved by intubation.

The prognosis of tetany after thyroid operations varies with **the** amount of damage done, but is usually grave and often fatal.

Tetany of gastric dilatation is usually an indication of an **early** fatal termination unless the underlying condition can be relieved. In ordinary gastro-intestinal disturbances it passes away with the relief of the cause.

The prognosis of tetany of pregnancy is usually good, although fatal cases have occurred, and the trouble will recur in subsequent pregnancies.

Diagnosis.—The diagnosis of tetany is not difficult if we bear in mind the localization of the spasms, their bilateral character, and can demonstrate the presence of Trousseau's and Chvostek's signs and the increased irritability of motor and sensory nerves.

When tetany affects the entire musculature of the body, and especially when it follows an injury to the tongue as happened in one of the writer's cases, it becomes necessary to rule out tetanus. In the case just cited it was very difficult, because the masseter muscles were the first to become involved. The chief distinction lies in the intermittent character of the spasms of tetany, with the periods of complete remission, as opposed to the continuous character of the tonic spasms of tetanus. There is no opisthotonos in tetany, and whereas the involvement of the pharyngeal and respiratory muscles are the rule in tetanus, they form the exception in tetany. In tetany the first muscles involved are those of the hands and feet, and then the involvement ascends to the trunk; in tetanus the first muscles involved are those of the jaws and neck, and then the spread is downward, the respiratory, trunk, and abdominal next in turn; the muscles of the hands and feet are affected late and not to the same extent as in tetany. Moreover, the aspect of a tetanus case is that of a gravely ill patient, whereas the tetany patient may look and feel well.

There occurs in very young children a condition termed arthrogryposis (Henoch's idiopathic contractures). This consists of a tonic contraction of nearly all the muscles of the body, so that the child can be taken by the occiput and lifted up in one rigid mass, resting on the heels. This condition may last for weeks and months and end in recovery. The spasms affect mainly the trunk muscles. They are gradual in development, and are tonic, painless, and continuous. There is no Trousseau or Chvostek phenomenon, and no hyperexcitability of sensory and motor nerves.

It has been shown (Hochsinger, Gregor, Finkelstein, and Stoelzinger) that this condition of arthrogryposis is the result of defective artificial

feeding and disappears soon after mother's milk has been substituted for the artificial food.

Treatment.—The treatment of tetany must naturally be somewhat different for the different classes of cases. If it has been definitely established that hypoparathyroidism is the underlying factor, an attempt must be made along the lines of overcoming this defect. In the majority of instances, especially in the idiopathic cases, the function of the gland under ordinary conditions is sufficient, and only in the presence of an exciting cause in the form of some toxin, introduced from without or generated in the body, does this deficiency become manifest in the form of tetanic spasms. The indication, therefore, in the majority of cases is to find the source of the toxin and remove it if possible. Causal therapy is indicated, of course, in all cases, and this will be considered later on when the subject of postoperative tetany is taken up.

Tetany of Children.—Since robust children are rarely affected and the disease is found mainly in sickly, rachitic infants, suffering with gastro-intestinal disturbances, diarrhea, and emaciation, the first indication is to improve the digestion; strict hygienic regulations, fresh air, bathing, and proper clothing should be enforced. The gastro-intestinal indigestion calls for the regulation of feeding as a primary indication. This should be preceded by catharsis in the form of castor oil, perhaps very small doses of calomel, and then the administration of bismuth subnitrate or subgallate. Intestinal antiseptics, sulpho-carbolate of zinc, betanaphthol, benzosol, carbonate of guaiacol may be necessary before the intestinal fermentation can be checked. The colon should be flushed with warm sterilized water daily if the stools are offensive and large quantities of water given by mouth. The food should all be Pasteurized and food rich in calcium salts should be given. Stoelzner's contention that milk increases the tetany manifestations has not been borne out by others. Milk and its derivatives and eggs (foods which naturally contain calcium) should be given. If cows' milk is not well digested, one of the calcium salts should be added to the other foods. Tonics in the form of iron, quinine, and arsenic and especially cod-liver oil should be administered; when possible, wet-nursing should be substituted in the severe cases for artificial feeding. Recent investigations would indicate that the intake of sodium chloride should be restricted. Rosenstern found in infants with spasmophilia and gastro-intestinal disturbances that one large dose of calcium (100 c.c. of CaCl_2 , 3 per cent. sol.) reduced the electrical hyperexcitability as well as the other tetany manifestations, whereas the administration of sodium chloride increased these manifestations.

Hochsinger and Kassowitz advise the administration of phosphorus.

Lukewarm water and hot wet packs to the affected extremities afford relief.

Aside from the treatment based upon the hypoparathyroid origin of the endemic and epidemic variety of tetany, there can be no other causal treatment, the active toxin in these cases not being known, it cannot be the subject of prophylaxis. Calcium salts and general

hygienic treatment afford relief in these cases. The use of the various parathyroid preparations in these cases is still a subject of dispute, whereas, there is almost a unanimous opinion as to the efficacy of the calcium salts. These salts should be given in the form of the acetate or lactate, in 5 per cent. solutions, and although the results are somewhat slower, the internal administration is just as efficacious as the intravenous or the subcutaneous.

The success obtained by Danielsen in transplantation of human parathyroids would point the indication in these cases of tetany in which the relapses are frequent or which have become chronic. The human parathyroids could be obtained immediately after death, either in accident or other appropriate cases. The value of parathyroid preparations obtained from lower animals is still in dispute. Pineles found that the use of parathyroid preparations obtained from the horse were absolutely useless in the experimental tetany in cats, whether they were administered dry or fresh, per os, subcutaneously, or by intraperitoneal transplantation. Frankl-Hochwart is inclined to side with this view of the inutility of ordinary parathyroid preparations. Danielsen found no relief in his case with all forms of parathyroid preparations, whereas relief was obtained very quickly after transplantation of human parathyroids. On the other hand, successes with parathyroid preparations have been reported by Marinesco, Visale, von Eiselsberg, and others. In some cases success was obtained by combining thyroid with parathyroid preparations.

Oppenheim and Romanoff each report a success in the treatment of chronic tetany by transplantations of animal thyroid gland.

The use of the calcium salts has made symptomatic treatment for the spasms less necessary than formerly. Bromides are usually sufficient, although chloral and even hyoscine-hydrobromate, morphine, and chloroform may be necessary for the relief of the severe pains caused by the spasms.

Rest, warmth, and prolonged warm baths are indicated in the more severe cases.

The laryngospasm and the attendant dyspnea may at times require intubation, although one case in the Cincinnati Hospital with all indications of laryngeal dyspnea died notwithstanding the intubation.

Those cases which result from gastric dilatation and gastroptosis require special treatment, varying with the underlying cause. If there be a stricture of the pylorus nothing short of a gastro-enterostomy will afford relief. In gastroptosis and atonic dilatation the stomach should be emptied, preferably by an emetic. If the stomach-tube must be used great care must be taken in effecting the lavage. When emptied the stomach should be rested and rectal feeding resorted to, large quantities of water must be given by the colon, and an excess of chloride of sodium avoided. Fleiner advocates the subcutaneous injections of 0.5 per cent. saline solutions twice a day, a pint at a time. He also advocates gastric resection or gastro-enterostomy in gastric dilatation as soon as there is a rapid loss of weight or a diminution

in the secretion of urine, notwithstanding the factors of rest, and adequate quantities of food and fluids are retained.

If the symptoms yield to rectal feeding, stomachic feeding can be gradually resumed. As a matter of course the calcium treatment should be instituted in these cases.

Postoperative Tetany.—*Prophylaxis.*—Since the cause of postoperative tetany has been found to be injury or removal of the parathyroids, the technique of struma operations has been changed. The superior thyroid arteries are ligated distal to the branch going to the parathyroids, and the operation leaves the capsule of the thyroid and the adjacent thyroid tissue of the lower lobe intact. Moreover the entire thyroid for obvious reasons is never removed. The result of this improved technique has been a disappearance of postoperative tetany. When, however, tetany does occur the first indication is the administration of calcium salts in the hope that the injury to the parathyroids is only temporary. Remarkable recoveries have been recorded after one or two administrations of calcium salts. If these salts fail the parathyroid preparations, fresh or dried glands, extracts or tablets, can be administered. The fresh glands are said by Lowenthal and Wiebrecht to be ten times more powerful than the tablets. At times a combination of the thyroid and parathyroid tablets seem to give better results. The value of these preparations is still a matter of dispute. In a recent case of Danielsen, all ordinary means were tried—calcium salts, fresh and dried parathyroids, thyroid and parathyroid tablets without any success. Two human parathyroid glands were then transplanted between the abdominal fascia and peritoneum with complete success.

The tetany of pregnancy is usually mild. It yields to a combination of the bromide and calcium treatment. Fatal cases, however, have been recorded, and when severe and the child is viable, the pregnancy should be terminated. This is, however, rarely called for. If the attacks occur during labor, and the ordinary use of chloroform does not give relief, the labor can be terminated by the use of the forceps. If the attacks are especially severe during lactation, weaning the child gives relief. On account of the tendency for tetany to recur pregnancy should be avoided in the future. This is not only proved by actual experience in pregnant women, but is supported by the animal experiment of Vasali and Generali, who showed that partially parathyroidectomized animals, who showed only mild tetany, developed tetanic attacks in each subsequent pregnancy and labor.

CHOREA

Synonyms.—Chorea; St. Vitus' dance; Sydenham's chorea.

Definition.—It is essential for a clear understanding to limit the term chorea to a disturbance of the nervous system, characterized by widespread muscular movements, involuntary and purposeless in character, acute in its course and limited in duration, and often dependent upon a demonstrable infection. Habit tics, hysteria, hereditary

chorea and myoclonus must not be considered under this head, they being essentially different.

Etiology.—The causative factors underlying the development of chorea are of prime importance in the consideration of the treatment.

In the vast majority of cases the prime etiological factor is a state of irritable weakness of the nervous system. This state of the nervous system may be inherited, or it may be caused by a bad state of nutrition during infancy, or early childhood. Quite a number of these children present the lymphatic constitution, adenoids with enlarged tonsils, hyperplasia of the lymphatic system, with a hypoplasia of the cardiovascular apparatus. All developing nervous systems are characterized by irritability and lack of inhibition. Emotions are quickly translated into motor acts, and some children have a state of muscular instability which might be termed natural chorea.

The opinion is gaining ground that chorea is an infectious neurosis. This opinion is based upon the frequent association of chorea with rheumatic affections of the joints or endocarditis. The chorea usually follows the acute affections of the joints or endocarditis, but occasionally the two conditions occur simultaneously, or the rheumatic joint affections may occur after the chorea has already been developed. As to the endocarditis, this is seen only in an acute condition, in the violent cases

Chorea may follow scarlatina (2 out of 112 cases), whooping cough, measles, la grippe, and other infectious diseases, but not very frequently.

75 per cent. of cases, following the best statistics of Thayer, Osler, and others, are still unaccounted for on the basis of a rheumatic infection. Basing an opinion on the great prevalency of tonsillitis among children and young adults, and its undoubted relationship to acute articular rheumatism, it is not impossible that the toxins of tonsillitis, secreted and absorbed from diseased tonsils may play a great role in the development of a large number of the mild cases of chorea in which there is neither a history of rheumatism nor evidences of organic heart trouble. Of the 112 office cases referred to above, 11, or about 10 per cent., gave a history of tonsillitis preceding the attack.

In quite a large percentage of cases no toxin can be demonstrated. It is the expression of all laryngologists, however, that when enlarged tonsils are removed in any case where there has been a previous history of tonsillitis, scarlatina, or diphtheria, pus is invariably found in the base of the tonsil.

In some cases we are perhaps dealing only with an exaggerated state of functional irritability and loss of inhibition of the nervous system, caused by extreme nervous exhaustion. Emotional excitement or fright is only an exciting cause. A close scrutiny will show that choreiform movement to a mild degree existed before the emotional shock. Out of 112 cases only 7 gave a history of a scare. Prolonged mental strain or emotions of a depressing character may play an important role as predisposing factors. Trauma at times occurs as a cause (1 in 112 cases).

Chorea of pregnancy is rather infrequent (2 in 112 cases). It occurs usually in young married women who have had chorea before. It usually occurs in the first pregnancy, and may recur in subsequent ones. It is seen especially in illegitimate pregnancy. Here, fear, worry, and a sense of disgrace are predisposing factors. It begins in the second or third month, and while it occasionally disappears in the course of two or three months it usually continues until delivery has taken place, or may even continue during lactation. We at times see Basedow's disease develop during pregnancy. Whether chorea can be caused by a toxin originating from a lack of compensatory balance of internal secretion during pregnancy or whether it is a reflex internal disturbance has not as yet been determined.

No definite conclusions have been arrived at as to the bacterial cause underlying the infectious agent at work in chorea.

Pathology.—A disease which terminates in the vast majority of cases favorably cannot have any gross destructive lesions as its pathological basis. Even the paralytic cases recover completely, and we are therefore justified in saying that the toxin produces only a functional and temporary change in the ganglionic cells involved. It has always been held that the seat of the affection is in the cortex of the cerebral hemispheres, but this view has more recently been brought in question.

We can conclude, therefore, that the gross lesions, such as hyperemia, embolic or thrombotic areas of softening in the cortex or basal ganglia, minute hemorrhages, etc., which are found in fatal cases are secondary to the endocarditis and the septicemia, and that there are no gross lesions either in the brain or cord in the ordinary cases of chorea minor.

Symptomatology.—**Mild Cases.**—The vast majority of cases of chorea are of the mild type. There may be a prodromal period during which the child is fatigued easily, is irritable, inclined to cry easily, to be stubborn and hard to manage. The digestion may be poor and the sleep restless. Then begin the involuntary, incoördinate movements. They usually begin in one hand or arm, then perhaps attack the leg on the same side or the opposite arm. At the same time the muscles of the face become involved, movements of the mouth, jaw, and orbicularis, the making of grimaces. Gradually the movements increase in severity and extension, and the patient is in a constant state of activity. The movements are involuntary and without purpose, and are increased by emotions, and especially by embarrassment. Sometimes the movements are more marked on one side, and then we have the so-called hemichorea. The tongue cannot be held in a protruded position, nor can the child remain quiet on command; the head is twisted from side to side; there is a constant play of the fingers, and a tugging at the garments, flexion and extension of the arms, shrugging of the shoulders.

As a rule, the motor unrest does not affect the eye muscles, although there may be an inability to fix an object.

Speech is affected even in mild cases. The children show a disinclination to speak on account of a conviction that they cannot speak properly.

The movements are never rhythmic nor spasmodic; they are constantly shifting from one group to another; arms, face, leg, and trunk muscles are affected irregularly and in constant variation.

Voluntary movements can be executed, but are often interfered with by the intervention of the involuntary. Writing and manual work usually suffer for these reasons, and whatever movements are necessary are executed in a hasty abrupt manner. Walking is usually possible, although the gait is awkward.

The movements usually cease during sleep, although Oppenheim and others have described cases of chorea nocturna, in which the movements were most marked during sleep.

Severe Cases.—These cases are usually unable to walk on account of the great degree of motor unrest, but are mentally still in a normal state. There is an exaggeration of all the movements described above. The body may show many bruises caused by awkwardness and falls. Self-feeding, which in the mild cases was possible, excepting a few mishaps with fork and spoon, is now impossible. Mastication and swallowing become difficult on account of the constant movements of the tongue and jaw and pharyngeal muscles.

Respiration is irregular and jerking. Speech becomes very much disturbed, and mutism is often seen. The involvement of respiratory and laryngeal muscles often gives rise to a tendency to emit inarticulate noises. Motoric unrest and incoördination reaches such a degree as to render the patient unable to leave bed, and renders padding of the bed necessary. Sleep is much disturbed, emaciation rapidly sets in. Fever is usually present and acute endocarditis a concomitant condition. This latter state, either occurring as a severe case from the start, or rapidly increasing in intensity, has been called *chorea insaniens*. The enormous motor unrest (*Folie musculaire*), the unintelligible sounds emitted at irregular intervals, the inability of the patient to speak, the anxious facial expression, constantly changed by grimaces, give the case an appearance of insanity, which is rarely real, usually only apparent. In a few cases there may be real delirium, but it is questionable whether this is not caused by the septic fever rather than the chorea itself.

Psychic Changes.—In the mild cases of chorea we usually see changes of character, rarely distinct mental alienation. In adults the latter condition occurs, but very rarely. Inattention, lack of concentration, irritability, and emotional instability are usually present. The children become self-willed, disobedient, and rebel against restraint and authority. In the more severe cases the vacant facial expression denotes the mental and emotional sluggishness. At times night terrors, visual and auditory hallucinations occur.

Acute delirium may occur in the cases which terminate fatally. If these latter cases recover, they may have a condition of stupor and acute dementia, from which the recovery may not be complete.

Oppenheim states that adults, more especially during pregnancy, may develop a real psychosis, under the picture of acute hallucinatory

delirium, with intercurrent maniacal states, lasting for weeks, followed by a state of mental depression, ending either in recovery or death.

In a few cases, distinct hysterical complications are seen, manifesting themselves in the form of echolalia or coprolalia.

The reflexes are usually increased, but in a few instances may be absent. When they are absent it has been attributed to hypotonia, although the writer has never seen this condition.

Paralytic Phenomena.—Church states that the motor power is diminished by 40 to 50 per cent. It would be better to say that motor power is in abeyance rather than that real paralysis occurs. The child is brought with the statement that one side of the body is paralyzed, the arm hangs limply, and the leg of the same side is dragged. The child, however, walks, and, on command, can execute all movements with the affected extremities. Other cases present a picture of paretic ataxia which renders them hardly able to walk. This condition can attain such a degree that the patient lies in bed as if paralyzed, the upper extremities and face may show choreic movements, but they can be detected only with difficulty in the involved extremities (chorea mollis, limp chorea, paralytic chorea). The muscle tone may be lowered and the reflexes diminished; this condition may follow or precede choreic movements; the muscles, however, usually retain their normal volume, and the electrical excitability remains unchanged. An examination of the affected extremities fails to show any evidence of the organic origin of the weakness; there is no rigidity and no ankle clonus or Babinski sign. The pseudoparalysis disappears with the subsidence of the choreic movements elsewhere, and its origin may be due to the influence of the toxins on the cord cells, although in some of these cases the writer is convinced that the paresis was due to autosuggestion.

Course, Duration, and Prognosis.—There does not seem to be much difference in duration between mild and grave cases of chorea. The average duration in the writer's experience has been between two and three months. The fatal cases seem to run a much shorter course, usually between two and three weeks. Cases, however, may become chronic, lasting even several years. The tendency to relapse is marked.

The prognosis is more grave in the chorea of pregnancy. While occasionally the chorea of pregnancy may run its course in two or three months, it usually persists throughout pregnancy, and usually terminates soon after delivery, although it may persist during lactation. The mortality during pregnancy is as high as 25 per cent. The total mortality is not over 2 per cent.

Treatment.—Prophylactic.—Many children have almost a natural chorea. Inhibition is at a very low ebb in most modern children, both in the tenements and in the pampered children. Emotion and desire are almost instantly transferred into muscular activity. The children are not only active, but irritable, restless, fretful under restraint, and unremitting in their endeavor to carry out all and especially forbidden

acts. These children are usually tall, thin of skin, narrow and long of chest, long-legged with poorly developed muscular and osseous systems. In addition such children are of lymphatic constitution with adenoids, large tonsils and, perhaps, a hypocardiovascular system. These children are prone to the development of infectious diseases and more especially tonsillitis. The prophylaxis consists in curbing the natural tendencies to restlessness in these children by keeping them within the narrow confines of a simple child life. The first requisite for each child is a long night's rest in bed, not less than ten to twelve hours, with an hour or two in bed at noon. They should have daily spray baths, just a dash of hot and cold water while standing in two or three inches of warm water. The meals should be very simple, milk, cocoa, bread, cereals, fruits, thick vegetable soup, fresh vegetables; and the meals arranged so that they may be at least four hours apart, with only milk or fruit between meals. The last meal of stewed fruit, milk, and bread should be given at 5.30 P.M., so that the child can be in bed not later than 7 P.M.

The motor activity and restlessness can have an outlet in out-door play, or if the child is over four or five years old in nature schools rather than kindergartens. Nature schools are preferable, because they entail outdoor life all the year round; the interests are sustained better by inquiry into the traits of plants, trees, flowers, birds, and animals rather than by the systematic routine play and walk in the kindergarten.

These children are all very apt at learning, and whenever possible should have some private tutoring, and should not enter on the regular routine of school until the ninth or tenth year.

The systematic removal of adenoids and enlarged tonsils, especially if there has already been an attack of tonsillitis, is the most important of the prophylactic measures. It is hardly necessary to call attention to the relation between tonsillitis and rheumatism. But aside from the size of tonsils interfering with proper sleep, and the proper oxygenation of the blood, it is the constant experience that the base of the tonsil which has been the seat of an infection always contains pockets of pus. We have therefore from enlarged tonsils a constant absorption of toxins, and if these latter are not the actual cause of chorea, they, combined with the mechanical disturbances of enlarged tonsils above referred to, produce anemia and general nervousness and render the child an easy victim to the infection which actually causes the chorea. The tonsils should be removed radically, for the tonsillotome merely removes the redundant top and leaves behind the infected base.

General Treatment.—When the chorea is actually developed the course of treatment varies with the grade of severity of the case. The child if it is old enough to go to school should be kept at home. As a matter of course all sources of emotional excitement should be removed. All tendency to correct or punish the child for its wilfulness or disobedience should be warned against.

The ordinary mild case with an absence of fever and no evidence

of an acute endocarditis is treated better by a combination of rest and out-door life by being put to bed. The child usually rebels against confinement in bed on account of the mental and physical restlessness, and unless a skilful and resourceful nurse can be employed I have found that the following regime is best in the great majority of cases: The bath should be given at 7 A.M., either a sponge or a spray bath, one minute hot, one minute cold, preferably in a bath tub with three or four inches of hot water in which the child can stand. In cool or cold weather the room should be warm. The child is then put to bed again and given a breakfast at 7.30 A.M. It is allowed to remain in bed until 9 A.M., and then dressed and sent out of doors, either to walk or perhaps play with one or two other children. At 10.30 a glass of milk is given, at 12.30 dinner, and at 1 o'clock the child is undressed and put to bed for two hours. The afternoon is spent out of doors if possible. Supper is given at 5.30. At 7 a prolonged warm tub bath (90° or 100°) for five or ten minutes, or a warm sponge bath for two or three minutes is given; the child is then put to bed.

The diet should be simple and plain. No coffee, tea, or alcohol is allowable. Usually it is best to exclude beef and an excess of salt from the diet.

In the more severe cases the poor should be sent to hospitals and the well-to-do put to bed at home in charge of a trained nurse. Strict isolation is not necessary unless there is an hysterical element. When in bed all the physical adjuncts of a rest-cure should be given, viz., massage, hot packs for fifteen to twenty minutes if the restlessness will allow it, spray baths, active and passive movements, and alcohol rubs at night. It is needless to say that proper protection must be taken to prevent the child in the restlessness from falling out of bed or from bruising itself.

It is very important to correct any tendency to intestinal indigestion which manifests itself by distention of the stomach and intestines with gas. This is often the result of irregularity in the time of eating meals, and is often corrected by having an interval of four hours or more between meals, with nothing but fruit in the intervals. If this does not suffice it will be necessary to eliminate all of the starches from the diet list, excluding potatoes, sweet potatoes, carrots, turnips, peas, dried beans, white bread, breakfast foods, pastry, pies, pudding, cake, and soft candies.

In the severe cases, especially when fever and rheumatic joint affections are a complication, free saline catharsis with small doses of calomel and sodii benzoate should be given on the first day to be followed at once with doses of sodium salicylate (freshly precipitated) of sufficient size and frequency to produce the physiological effect. Aspirin and novaspirin can be used, but salophen, antipyrine, and all coal-tar derivatives had better be avoided on account of the possibility of an infectious myocarditis being present, even though signs of an endocarditis are not manifest. Brilliant results, however, have been reported in very acute cases as a result of the use of large doses of antipyrine. In

children, from 3 to 5 grains, in adults 15 grains every two or three hours, have been given with good results.

In ambulant cases a combination of iron and arsenic should be given either separately or combined in such a manner that the arsenic can be pushed in the course of two or three weeks until the eyes become puffed, or colicky pains and looseness of the bowels result. Mere puffing of the eyelids in the morning should not cause a discontinuance of the arsenic. When the maximum dose which the individual can tolerate has been reached, this dose should be continued for at least four weeks, and then the dose reduced in the same gradual way as it was increased. Although iron and arsenic seem incompatible I have for years combined Fowler's solution with the albuminate of iron:

R—Sol. Fowlerii	20.0
Liq. ferri album., Drees	40.0—M.

Sig.—Begin with 10 drops t. i. d., after meals.

Take one drop more each day until dose reaches 25 to 30 drops. In this manner the Fowler's solution is increased by $\frac{1}{2}$ drop each day.

Arsenious acid may be given in dose varying from gr. $\frac{1}{150}$ to $\frac{1}{60}$ (0.0005 to 0.001), varying with the age, and can be combined in pill form with some preparation of iron. This combination has the disadvantage of an impossibility of the gradual increase of the arsenic. In older individuals the cacodylate of sodium can be given intramuscularly, at first daily for six or seven days, then every second day until twenty-four doses are given. The dose should vary with the age, from 0.03 to 0.1 gram or $\frac{1}{2}$ to 2 grains. This preparation of arsenic is more appropriate for the severer cases or for chorea insaniens. It is less apt to produce signs of arsenical intoxication than arsenical preparations which are administered by the mouth.

Sedatives are nearly always indicated, and except for the chorea insaniens, the bromide preparations usually suffice. The bromides are rarely indicated during the day and had best be avoided on account of their tendency to disturb the digestion. The bromide of strontium is the least harmful, and it should be given at bedtime or three or four hours after supper, in doses varying, for age, from 10 to 40 grs. (0.6 to 3.0). Even in the more violent cases the bromides are preferable to chloral, on account of the depressing influence of the latter drug on the heart. Oppenheim has seen good results in very bad cases of chorea insaniens from the use of the hypodermic injections of morphine. In a disease, however, in which infection plays such a prominent role all opiates had better be avoided, except as a last resource, and then special attention must be paid to intestinal elimination. In these cases of chorea insaniens hyoscine hydrobromate is indicated, but must be used with great care, and in very small doses. All the other sedatives have been used, chloralamid, camphor monobromate, sulphonal, trional, valerianate of zinc, and brilliant results have been obtained. Apomorphine has been found to be useful in the treatment of the very severe cases. Electricity is superfluous and of doubtful value.

In prolonged cases the method of reëducation of Meige and Feindel, the inhibition therapy of Oppenheim, and the respiratory exercises of Pitres can be resorted to. For a description of these methods see treatment of tic convulsive.

Hypnotism is certainly not indicated in the milder cases, and in the severer cases it cannot be successfully carried out. It is questionable whether the successes reported by this measure were in cases of acute chorea of an infectious type. Chorea insaniens, of course, demands bed treatment; the bed should be boxed in, and padded, and the patient given the freedom of a large bed without mechanical restraint. In cases with fever, it is advisable after free catharsis with calomel and salts, to institute the sodii salicylate treatment in large doses. This should be continued for three or four days, or even a week before it is given up as useless. After the salicylates have failed, the cacodylate of sodium should be used as described above. The hypodermic use of hyoscine hydrobromate or morphine may be necessary, but before these are resorted to, a combination of chloral and bromide should be given every two or three hours. The other sedatives described above can be used if these fail. The chief indication in these cases is the feeding, and on account of the great difficulty of mastication the diet should be restricted to milk and raw eggs and other liquid foods with an abundance of water.

The chorea of pregnancy requires special consideration. On account of the danger of injuring the unborn child, it is best to confine the use of sedatives to the bromide of strontium, and to be careful in the administration of arsenic, avoiding the maximal doses. The use of the cacodylate of sodium hypodermically in one grain doses in the chorea of pregnancy had no bad effect on the course of the pregnancy in a recent case treated by the writer. The question of bringing on labor in the very violent cases often has to be considered. This may be necessary on account of the violence of the movements, the acuteness of the exhaustion, the accentuation of or danger from a heart lesion, the presence of nephritis, or the onset of an acute psychosis.

In the writer's opinion, this ancient ethical question can only be answered in the affirmative, when the pregnancy has advanced to the stage at which a viable child can be born. It must be borne in mind that it is not infrequent for severe violent cases of chorea, even chorea insaniens to run the ordinary course in the early months of pregnancy and terminate in recovery in two or three months without interfering with the pregnancy.

THOMSEN'S DISEASE (MYOTONIA CONGENITA)

Etiology.—The most pronounced factor in causation is heredity; usually several members of the same family are affected at the same time. It can be traced either in the direct line or in collateral lines.

Blood relationship (Bernhardt) between the parents has been known to originate the disease in the offspring. In some cases (myotonia

acquisita) no hereditary predisposition could be established. In some families there is a history of neuropathic or psychopathic degeneracy.

The disease may originate in infancy. In childhood, the children are awkward, stiff, and cannot play out-door games well. It most frequently becomes very manifest about the age of twenty. Sometimes fright has been known to be the immediate cause, but it is held that this factor brings into active manifestation a condition which was either latent or so mild that its symptoms were misinterpreted and the individual was regarded as being merely awkward and clumsy.

Pathology.—The basis of the disease is perhaps a congenitally defective condition of the neuromuscular end-apparatus. Knoblauch holds that in myotonia we have the antithesis of myasthenia. In the latter disease the congenital defect in the formation of the muscles shows itself in a predominance and pathological excess of the pale, striated muscle fibers; in myotonia there is an excess of the red striated; the defect is an embryological one.

The fact that a number of cases of myotonia develop atrophies lends color to the assumption that myotonia is related to the dystrophies, and perhaps this form is the connecting link between the two. Some of the histological examinations show the presence of some muscle fibers in a more or less advanced state of atrophy, side by side, with hypertrophic fibers (Hoffman, Pels, Koch, Frohman).

Our knowledge of the pathology is limited to the study of muscle tissue excised during life. Most of the investigations seem to indicate that we are dealing with a primary disease of the muscles.

Most writers today hold the myopathic theory of the origin of this disease. We have, perhaps, according to Knoblauch's theory, a congenitally defective organization of the muscle tissue, and as an exciting cause a toxin which results from a disease of metabolism.

Voss has lately proposed the theory that myotonia is nothing but a modification of the spasm of tetany, because of the fact that tetany and myotonia are often seen combined in the same individual. The value of this suggestion is that myotonia, like tetany, may be due to a deficiency of parathyroid action, and that the disorder of metabolism may yield to the same treatment, viz., the calcium salts. Similar observations in tetany have been made by Frankl-Hochwart, Karporek, Bettman, and Hoffman; but Frankl-Hochwart rather denies that the spasms seen in tetany should be classed with myotonia.

Symptoms.—Myotonia is a disease in which voluntary contraction of muscles brings on spasms of the group whose use is attempted. These spasms inhibit all movements for the time being or make the movements awkward and clumsy. The muscles affected appear hypertrophied and firm, but the muscular power evolved is less than normal. One of the striking characteristics of the disease is the apparently powerful development of the muscles in contrast to their weakness; although (Mann) the weakness which is present on beginning use of the muscle is said to disappear with exercise. The hypertrophy is more marked in the muscles which are mostly affected by the disease.

In infancy, the disease may manifest itself by defective use of the tongue and lips in nursing, or by the sudden rigidity of the face during crying.

Older children are usually awkward and clumsy and cannot take part in the outdoor play of children. The chief clinical manifestation is the occurrence of tonic spasms in muscles whose use is attempted. These spasms are especially apt to occur when a quick voluntary movement is attempted, such as making a fist, flexing the arm on the forearm, or sudden closing of the mouth. The spasms cannot be relaxed by the will, but after a variable period of time, from five to thirty seconds, relaxation sets in. The spasm is repeated each time a muscular effort is attempted, but each succeeding spasm is less violent and less prolonged. Finally if the voluntary effort is persisted in, the spasm ceases and the intended movement can be accomplished, but a change in the rhythm of the muscular action or the rapidity is accompanied by a new series of spasms. Thus, on arising from a chair, the movement of the legs is hindered at once by a spasm of the muscles of the legs and thighs, as the spasm relaxes, the attempt to walk is immediately followed by a second spasm, but as the attempt to walk is persisted in the spasm gradually dies out and the patient is able to walk long distances, or to dance without any inconvenience; after a rest the same phenomena are again observed; a halt, or sharp turn, or a change in the rapidity of the gait may bring on a new series of spasms. Passive movements do not bring on the spasm.

An attempt to elicit reflexes will bring on a spasm in the contracting muscles, and thus change the nature of the reflex contraction. The spasms are never painful, an energetic reflex movement, such as sneezing and coughing, or a sudden start as a result of fright, may bring on such a widespread tonic spasm, as to cause the individual to fall to the ground, rigid, stiff, incapable of any movement, until the muscles relax.

Fatigue, hunger, extremes of cold and heat, mental depression all increase the violence of the spastic contractions. Mental quietude, moderate use of alcohol, hot tea, moderate exercise, and warmth diminish the spasms.

These muscular spasms are characterized by their slowness, by their tonic character, and by the continuance of the muscular contraction after the voluntary impulse originating it has ceased. All of the voluntary muscles of the body may be affected, including the orbicularis palpebrarum, the external eye muscles, and the muscles of mastication. The muscles involved in vital functions, such as swallowing, respiration, micturition, defecation, and parturition, do not seem to be affected.

The disease affects principally the lower extremities. It may be more marked in one group of muscles, and only slightly in others. It may remain restricted to only a small group of muscles or even a single group. Thus, Oppenheim reports a case limited to the orbicularis palpebrarum. At times the upper extremities are more involved than the lower, but this is the exception.

Paramyotonia Congenita.—Eulenburg and Bernhard have described a form of myotonia which affects symmetrical groups of muscles, and in which the myotonic spasms can be brought on by exposure to cold. The muscles affected are those of the face, neck, deglutition, and extremities. The spasm may last as long as fifteen minutes and render the individual helpless. After the spasm passes away the muscles are paretic, and the weakness may last for hours or even days.

Myotonia *acquisita* is a form of this trouble which shows various symptoms of myotonia in individuals who have no family history of the disease. The cause in these cases is attributed to injury or to overstrain. The spasm occurs partly when the muscles are at rest, and appear especially after exhaustive use of the muscles. This form seems more favorable; the manifestations are of an intermittent character and seem curable. Many of these cases are so mild as to cause little inconvenience. Others show an absence of the myotonic reaction of the muscles.

Diagnosis.—The diagnosis is easy in typical cases. The arrest of voluntary movements by the occurrence of spasms of a tonic character, and the presence of the myotonic reaction in the muscles, occurs only in Thomsen's disease.

Prognosis.—The prognosis as to cure is bad, although intermissions occur at times. The disease lasts throughout the life of the patient, but is not progressive in character.

Treatment.—If the disease is fundamentally due to an embryological maldevelopment of the muscles there can be no hope of effecting a cure. The fact, however, that its manifestations are mild early in life, and that there is a progressive increase in the severity of the symptoms up to a certain age, and that the first manifestations of the disease do not occur in some cases until adult life is reached, leaves some hope that an exciting cause in the form of a general disorder of metabolism may be present, which may be combated. If the contention of Bechterew is true, that there is present a gouty diathesis, the indications for treatment along these lines should be given a fair trial. Voss' observations of the relationship between myoclonia and tetany deserve attention. Spasms occur in both disorders, and the difference in the form of the spasms and the manner of their occurrence might be due to structural differences in the muscles. If the toxic element is due to a disordered metabolism caused by hypoparathyroidism the calcium treatment as outlined in the chapter on Tetany should be instituted; at the same time, there should be a preponderance in the diet of those foods, like milk and eggs, which are rich in calcium salts.

Thyroid and orchitic extracts have been used without results. Basing his treatment on the theory that the hyperexcitability of the muscles was caused by deformity or malformation of the terminal end-plates in the muscles, Gessler attempted in two cases to cause a complete degeneration of these end-plates by stretching the nerve trunks in the lower extremities, and reported favorable results as far as an improvement in the spasms was concerned. Both of Gessler's cases were later

on critically examined by Leiffer, and he found that the improvement was only a temporary one, but that on the contrary the patients were inconvenienced and damaged by the resulting paresis of the muscles and the development of sensory defects. The attempt at nerve stretching has not been repeated.

Mild gymnastic treatment and massage produce an improvement, moderate regular exercise, walking for instance, is found to be beneficial.

Hydrotherapy has also been advocated. The internal administration of tonics and the iodide of sodium may be of some value. In a prophylactic way the patient should avoid exposure to cold by dressing properly and should strive as far as possible to avoid emotional disturbances and excessive fatigue.

HUNTINGTON'S CHOREA

Synonyms. — Chorea chronica progressiva; chorea hereditaria; dementia choreica.

Etiology. — This disease is distinctly a family affection, and is inherited alike by the male and female members. Some reporters assert that when it skips a generation the disease dies out in that branch of the family.

The disease usually comes on in adult life, between thirty and forty years of age. Heilbronner states that the disease shows a tendency to develop later in life in succeeding generations.

Symptomatology. — The symptoms present a combination of motor unrest, associated with slowly progressing mental deterioration, terminating in dementia.

The onset of the motor unrest is gradual, beginning in the face and hands, increasing in extension and intensity as the years pass by, until all voluntary muscles are affected. The movements on the whole resemble those of acute chorea. The movements are involuntary, purposeless, and incoördinated; the face, arms, trunk, and legs are in a state of constant movement; the face is drawn up on one side, the shoulders elevated, the fingers flexed and extended, the trunk twisted, the legs jerked about, there is constant grimacing and incoördinate gesticulation. The eye muscles are usually not affected. Speech is interfered with by the movements of lips and tongue. The gait becomes uncertain and unsteady, due to jerking movements of the legs. The movements are all increased by emotional disturbances. They usually cease during sleep. Volition exerts a calming influence, and patients can suppress the motor unrest sufficiently to dress and feed themselves, except in the advanced stages. Intention movements have a temporary inhibiting effect on the choreic movements.

Walking is possible even in the advanced stages, but the gait becomes unsteady and often grotesque, as a result of the sudden and quickly changing irregular and unexpected movements of the trunk, hip, and

legs. Muscular power usually remains normal. In the writer's case there was a paresis of the left facial region. In one case a hemiparesis was noted. The reflexes are usually exaggerated. In the writer's case they were absent. Sensation is normal.

In addition to the motor phenomena, mental changes are invariably noted. These usually begin after the choreic movements have been in existence for some years. In the beginning they take the form of apathy, indifference, and lack of concentration. Often there is added marked mental depression, alternating with irritability and even passing conditions of acute excitement. Throughout the course of the disease, there develops a gradually increasing mental weakness which in many cases terminates in dementia.

Prognosis.—The prognosis is bad, the disease is progressive, and its duration varies from eight to thirty years. If the patient does not die of an intercurrent disease, he finally becomes bedfast, and dies of malnutrition, or in the state of coma superinduced by repeated attacks of pachymeningitis interna hæmorrhagica. Some cases do not pass into the state of terminal dementia.

Treatment.—There is but one ray of hope in the treatment of chronic chorea, and that is the close resemblance of the pathological findings to those of dementia paralytica. There is a possibility that the hereditary role may be played by an attenuated form of syphilis, and that a mild continuous use of antisyphilitic treatment in the form of mercury, iodide of potassium, and the intermuscular injections of cacodylate of sodium may be of some use. In a disease as hopeless in its therapy as this one is there can be no serious contraindication to the intravenous administration of salvarsan. Sedatives are always indicated, and the movements may be ameliorated by the use of bromide of strontium, given in 30 to 40 grain doses at bedtime. The general hygiene should be the same as that of acute chorea.

CHOREA ELECTRICA

Under this heading various conditions have been described which are partly of an infectious character, partly hysterical, and in part bearing a resemblance to myoclonia.

Dubini's Disease.—This obscure disease was first described by Dubini in 1845. It bears no resemblance whatever either to acute or chronic chorea, but has derived the name of electric chorea from the quick jerking movements which are present in the face and extremities.

Etiology.—The disease occurs in and seems confined to Northern Italy, and especially Lombardy. The cause is unknown, although infection seems to play the chief role. It can occur at any age.

Pathology.—The pathological findings are those of an infectious cerebrospinal meningitis. There is no pus formation, the meninges are inflamed, the cerebrospinal fluid is increased, areas of softening in the cortex and basal ganglia are found together with splenic enlargement and pulmonary congestion.

Symptomatology.—The onset of the movements is usually abrupt, preceded by pains in the head, neck, and back. The movements are quick, lightning-like, as if produced by an electric current. The spasms are frequent and affect an arm or a leg or the side of the face, first on one side and then involving the other. Epileptoid seizures occur, at times general, at others unilateral. The seizures occur without loss of consciousness, and are usually followed by paresis. Paresis develops in the course of time, usually beginning in the extremities, which were the seat of the first spasms. The paresis becomes more or less general, and atrophy and disturbances of electric reaction are present. The disease runs an acute or subacute course. It may terminate in a week, or last as long as five months. Sensation is affected in the sense of hyperesthesia. Slight touch is capable of producing seizures; the spasms are painful, consciousness is not impaired, fever is usually present.

The disease progressively becomes worse, the spasms become more generalized and more intense, the epileptic seizures become more and more frequent, at times a number in one day. The fatal termination is preceded by more or less continuous epileptic seizures and coma. A fatal termination occurs in 90 per cent. of cases, the cause of death being often cardiac paralysis. When recovery takes place, it seems to be spontaneous, and not the result of any special treatment.

Treatment.—No form of treatment has been found to be of much avail, in the absence of any known cause only symptomatic treatment can be instituted, this should consist of free purgation with calomel and salts. The salicylates and sodium benzoate in large doses would be indicated to overcome any infectious agent. For relief, chloral and bromides should be given.

Henoch's and Bergeron's Electric Chorea.—*Henoch's electric chorea* usually affects young children. The spasms are quick and violent as if produced by an electric shock. They occur at intervals of from three to five minutes, and usually affect the muscles of the neck and shoulders. Most writers are agreed, that this disease, which bears no resemblance at all to acute chorea, should be classed among the myoclonias.

Bergeron's electric chorea affects older children, usually up to the age of thirteen or fourteen years. It is characterized by the suddenness of its onset, and the rapidity with which it attains its maximum. It occurs usually in anemic and irritably nervous children, and the attack seems to bear a relation to gastric disturbances. The spasmodic contractions resemble contractions of groups of muscles produced by strong electric currents. They are sharp, strong, and brief. The head may be thrown from one side to the other, the shoulders suddenly elevated, the forearms violently flexed, the arm abducted. The spasms may remain localized to one part of the body, but they are usually generalized. They cease during sleep. Voluntary efforts at control not only fail, but often intensify the spasms. Sensation is normal. The prognosis is good, and the disease usually terminates after a few days or weeks in recovery.

Treatment.—The prevailing opinion of the French, among whom Bergeron's chorea is seen most frequently, is that the disturbance is secondary to gastric disorders. The fact that relief is frequently obtained by the use of emetics is an evidence of the psychic origin of the manifestations. Emetics should always be given and, if necessary, repeated.

In severe cases the patients should be strictly isolated and the regimens of the rest cure with cold spray baths, massage, packs, and active and passive movements, carried out. Arsenic is of undoubted value.

The Myoclonias: Paramyoclonus Multiplex.—**Etiology.**—The cause of this disease is unknown. In some cases there has been the history of an infectious disease, in others fright or trauma has been looked upon as the cause. The French writers Raymond, Brissaud, and Meige claim that myoclonias develop on a basis of neurotic or psychopathic degeneracy.

Symptoms.—Paramyoclonus multiplex is a functional neurosis characterized by clonic muscular twitchings which occur rapidly and often rhythmically at irregular intervals. The spasms may affect a part of a muscle, a whole muscle, or a group of muscles. They usually occur bilaterally in symmetrically situated muscles and may affect the trunk or the extremities. The muscles of the face are affected slightly or not at all. Muscles, like the supinator longus, which cannot be contracted alone by the will, may be the seat of these spasms. While all the muscles of the body may be affected, the muscles usually involved are the biceps, supinator longus, cucullaris, quadriceps femoris, and the semitendinosus. The muscles of the trunk and those immediately attached to the trunk are usually affected, while the muscles of the forearm and hand and the muscles below the knee-joint usually escape. Cases involving the face and the diaphragm have been reported. The neck muscles are frequently involved. In one of the writer's cases the contractions were limited to the muscles of the abdominal wall, in the other to the abdominal, gluteal, and thigh muscles.

The spasms usually occur in series, are usually severe, follow each other quickly, and in great number. During the interval the muscle may be at rest, or there may persist a fascicular twitching in small bundles of the muscles. The muscles involved are usually bilateral and symmetrical, but the contractions are not necessarily synchronous, even though both muscles are affected at the same time. While the contractions are quick, sharp, and violent the shortening of the muscle is rarely of such a character as to cause any movement in the sense of flexion and extension. Often no movement whatever can be detected in the affected limb.

The spasm is clonic in the typical cases, and may be repeated from forty to one hundred and sixty times per minute, and may continue up to ten minutes or more at a time, to be followed by a period of rest, varying from a half-hour to a day or more. The spasms may not occur in the symmetrically situated muscles at the same time, nor are all

the muscles affected simultaneously, but first one, then another may be the seat of the contractions; nor do the contractions occur at the same rate of speed in various muscles affected at the same time. In other words, the contractions themselves, occurring in various muscles at the same time are arrhythmic and unsymmetrical. They attack groups of muscles which cannot be thrown into contraction voluntarily; the movements show no coördination, and are of a type that cannot be produced voluntarily.

The spasms usually occur spontaneously, and are accentuated by fatigue; they cease during sleep, and are increased by strong emotions. Active voluntary movements have a quieting effect, as also does any occupation which absorbs the attention of the individual. Feré calls attention to the fact that position has a marked influence on the occurrence of the spasms. Lying in bed in a horizontal position increases the duration and severity of the attacks. In the sitting position the trunk is not affected by the spasms occurring in the neck and extremities. When standing the legs usually enjoy immunity, and walking reduces the spasms to a minimum, or they cease entirely.

Tapping the tendons of the affected muscles or even stroking the skin over them may bring on an attack.

These motor phenomena constitute the entire symptomatology. The motor power of the affected muscles is normal; there is no change in the mechanical or electrical excitability; the tendon and skin reflexes are usually exaggerated; sensation is normal.

Unverricht's Myoclonus Epilepsy.—Unverricht described a form of myoclonus which is combined with epilepsy, often terminating in dementia.

The myoclonia develops first, and in addition to the involvement of the usual muscles, the tongue, pharynx, and diaphragm are also involved. The epileptic seizures, at first infrequent, gradually increase in number.

The myoclonia is also progressive, finally becomes more or less constant, and interferes with the voluntary movements more and more.

The termination of this form of myoclonia in dementia has led Lundborg to draw a parallel with dementia præcox.

Prognosis.—The prognosis varies very much as to what the observer chooses to classify under this head. Starr says that the prognosis is good. The prognosis in the non-hysterical forms is very grave as to duration and remains subject to intermissions as a chronic disease. Numerous cures have been reported as a result of the use of electricity, but it is held that these were probably of the hysterical form.

Treatment.—It has been observed in animal experiments after the removal of the thyroid gland that muscular contractions similar to myoclonia develop, and for this reason the use of thyroid extract has been recommended. Thyroidin has also been used. Careful treatment with these remedies can do no harm, although the value of the treatment has not been established.

Lundborg advanced the theory that the spasms are the expression

of an auto-intoxication. If any associated disease is found causing either the production of toxins or defective elimination this should have proper treatment. Especial attention should be paid to the gastro-intestinal tract, and fermentation and decomposition of food be overcome by proper diet, habits of exercise, and the drinking of large quantities of water before meals, colon lavage, and the administration of intestinal antiseptics and occasional cathartics. The administration of arsenic, preferably in the form of intramuscular injections of cacodylate of sodium, will answer a twofold purpose, viz., general tonic effect of the arsenic, plus its action on the colon as an antiseptic agent. The cacodylate of sodium is administered by deep injection in the gluteal muscles in doses of 0.05 to 0.1 gram (1 to 2 grains) daily for six days, then every second day until twenty-four doses have been given.

As sedative agents the valerianates can be administered during the day, chloral and the bromides, preferably in the form of bromide of strontium, at bedtime; the former in 20 to 30 grain doses, the latter in 40 to 60 grains, varying with the severity of the attack.

Galvanism of the brain and spinal cord has been much lauded, and brilliant cures recorded. When used it should always be administered through a rheostat. The spinal electrodes should be placed over the nape of the neck and lumbar region for ten minutes.

Galvanism of the brain should be short in duration, not over one or two minutes, with one electrode over the frontal region and the other on the neck: the current should be weak, gradually introduced, and gradually withdrawn.

If there is an hysterical basis for the manifestations, psychotherapy will be indicated.

MYASTHENIA GRAVIS PSEUDOPARALYTICA

This is a general disease, affecting more conspicuously the muscles supplied by the cranial nerves, but involving all the muscles, even the unstriated, and therefore the term (suggested by Jolly) myasthenia gravis pseudoparalytica is perhaps the best name.

Etiology.—The opinion prevails that the presence of some toxin in the body is the exciting cause of this disease, acting upon muscle tissue congenitally defective. That this toxin must in the majority of cases be of endogenous origin is clear from the etiological fact that in most cases there is no history of infection.

That we are dealing with a combination of a congenitally defective neuromuscular apparatus, acted upon by a toxin developed in the body, seems probable. Whether, however, this toxin is due to a defect of the biochemical mechanism of the body caused by the function of a persistent or diseased thymus, or other lymphatic tissue, remains to be proved. Neuser, in his recent exhaustive monograph on the status thymico-lymphaticus, makes no mention of myasthenia as one of the clinical manifestations of this condition.

The researches of Pemberton on biochemical changes in myasthenia show an abnormal excretion of calcium salts and a deficiency of excretion of creatinin. He looks upon the disease as due to a defective metabolism of the muscle tissue and advises the use of the calcium salts in the treatment of the disease. All of the researches so far indicate that the pathological substratum of the affection is a disease of the muscular system of the body.

Symptomatology.—The essential manifestation of the disease is the rapidity with which the muscles exhaust when used. On arising in the morning, at least in the beginning of the illness, the patients are apparently normal; toward evening many if not all the muscles of the body are in a state of paresis. In the advanced cases, after a few voluntary muscular contractions, the power evolved decreases rapidly until in a short time the muscle is entirely exhausted. After a short period of rest the muscular power returns again. The earliest manifestations are on the part of the muscles supplied by the cranial nerves and the cases resemble the symptom-complex of progressive labio-glosso-pharyngeal paralysis.

Bulbar Symptoms—The first symptoms are usually on the part of the external muscles of the eyes. On arising in the morning the lids are in a normal position, as the day advances the lids gradually droop until at night the ptosis is well marked or even complete. After a night's rest the ptosis again disappears, but later on in the disease ptosis is also present on arising. Goldflam called attention to the fact that the external eye muscles are also involved in some cases. This shows itself in the form of diplopia, and during the examination the muscles fatigue and the images become separated farther and farther as the muscles become more and more exhausted. The images also show variation in position as the muscles become fatigued in turn. An irregular jerky movement of the eyeballs is seen as the eyes are moved frequently from side to side. The ptosis is at first compensated for by action of the frontalis muscles, and when these become fatigued the head is thrown backward. The involvement of the facial muscles gradually gives rise to a loss of expression, the lip muscles quickly show fatigue when used in speech, so also the tongue and pharynx muscles. Mastication and swallowing are attended with marked subjective symptoms of fatigue, and eating becomes difficult and finally almost impossible on account of the rapidity with which the muscles become exhausted. Speech becomes difficult and is attended with great exhaustion. In a few words we have ptosis of both eyelids, diplopia, dysarthria, dysphagia, paresis of the lip muscles, and weakness of mastication. Frequently the onset is characterized by a weakness of one or even of all the external muscles of the eye in the form of an ophthalmoplegia. The reflex action of the pupils is usually found to be normal.

These symptoms are all less manifest in the morning than in the evening, and may only be slight in the beginning of speech or at the onset of a meal and gradually progress until the speech becomes more

and more indistinct and mumbling, and mastication and swallowing become so difficult that tube-feeding must be resorted to.

The neck muscles become affected, and the head gradually sinks forward as the fatigue becomes more marked. The trunk muscles are involved. Dyspnea is a common symptom. In addition to the exhaustion, sudden attacks of severe dyspnea, with tachycardia and elevation of temperature, occur at times during the course of the disease. In one case at least, Strümpel was able to relieve this condition by pulling the tongue forward, and attributed the attacks to paralysis of this organ. The muscles of the extremities show the same involvement, the use of the arms becomes progressively more difficult; walking is attended with great fatigue and the patient finally becomes bedfast, although all movements can be executed in a recumbent position. In a word, the cardinal symptom of the disease is the rapidity of the onset of exhaustion after slight physiological use of the muscles. This condition is subject to remissions, disappearing quickly after rest, reappearing quickly on use, but in the progressive cases the paresis becomes to a greater or less degree permanent.

The second characteristic of the disease is that the signs of degenerative atrophy of the muscles are wanting, even to the end, in fatal cases. The muscles affected do not become atrophied, there is no fibrillary twitching, and no reactions of degeneration.

There are no sensory disturbances and no involvement of the rectum and bladder. There are no trophic changes.

Course, Duration, and Prognosis.—When the first articles were published, the disease was looked upon as progressive, with remissions and exacerbations, with an inevitably fatal termination. This view has changed. Its onset is gradual and chronically progressive, with acute or subacute exacerbations, followed by remissions, and may continue for as long as twenty years. The average duration in the fatal cases is from one and one-half to two years. Complete recoveries have been reported; whether these were not merely remissions of long duration cannot be determined. The prognosis however, is unfavorable, and nearly half of the reported cases were those which ended fatally. Death occurs as a result of exhaustion or of septic pneumonia. At times sudden attacks of dyspnea terminate fatally. The condition is capable of much improvement; even the most pronounced conditions of muscular weakness can disappear, and the patient present an appearance of perfect recovery, and then the paresis can suddenly and acutely reappear and quickly lead to a fatal termination. The remissions may last for nine years (Oppenheim), and Goldflam first called attention to the possibility of a cure.

Treatment.—If we hold the theory that the disease is the result of a combination of a congenital malformation of the muscle structure, plus the action of a toxin, which produces a degenerative atrophic myositis, it is clear that the only hope of therapy lies in our ability to find the source of origin of the toxin. If, again, we hold the thymus or defective lymphatic system theory of the origin of the lymphorrhagic

infiltration of the muscles as the cause of myasthenic gravis, we are confronted with an equally difficult problem in therapy. The indications for treatment with the toxic theory in view is offered by those cases which are combined with Basedow's disease and myxedema and has led Palmer to suggest the trial of thyreoidin and ovarian extract.

The treatment of the cases with thymus extract does not seem logical if we hold the theory that the persistence of the thymus gland and its secretions unbalances the effect of internal secretion on metabolism. The use of the x-ray for the purpose of destroying if possible any remnants of the thymus seems more logical and worthy of a trial. On account of the frequency with which disease of a persistent thymus has been found, careful use of the x-ray over the sternum should have a trial in all cases.

Chvostek's contention based upon certain resemblances to tetany, and of the opposite conditions of the state of muscle irritability, that myasthenia gravis is a symptom-complex caused by hyperfunction of the parathyroids, does not give any therapeutic indication. The use of anesthetics is not contraindicated in these cases if the theory is correct, that the disease is of muscular origin, and therefore, surgical treatment of the parathyroids either in the form of ligation of the branches of the artery going to the upper pair of parathyroids, or the removal of the latter could be attempted, although no cases have as yet been operated on with this theory as a basis.

There is no reason why tumors, even of the mediastinum, which may be the source of the toxin, should not be removed.

Electric stimulation of the muscles is contraindicated; the muscles will only be more exhausted, the clinical indication being physiological rest.

The chief indication for treatment afforded by a study of the nature of the disease is rest for the muscles. The patient is to be put to bed, all muscular activity, even speaking, is to be prohibited, and exercise is to be replaced by gentle massage and passive stroking of the muscles, and by passive movements.

The next indication is feeding. If the exhaustion of the masseter muscles is so great as to prevent mastication, the food should all be chopped fine, and given in a semisolid or liquid form, and great care should be taken to swallow slowly. As in true bulbar palsy the starchy foods are not well digested, because of insufficient mastication and mixture with saliva. The patient should keep starches in the mouth as long as possible, mix them with saliva, and thus prevent fermentation. Oppenheim warns against the use of the stomach-tube on account of the danger of sudden death during the struggle attending the introduction of the tube. Rectal feeding is necessary only when the patient cannot swallow.

In the advanced cases there should be periods of rest for the muscles of mastication and swallowing, during which these muscles are to be allowed to recuperate. Basing his experiments on the theory that the exciting cause of the symptoms of myasthenia is a toxin of exhaustion,

Weichard suggested the use of an antitoxin. Oppenheim, who has made use of this antitoxin, reports that he was unable to obtain any satisfactory results. Kauffman recommends the use of spermin (Poehl), 1 c.c. of a 2 per cent. solution, hypodermically daily, or every second day.

The hypophysis extract has also been used, but since there are no reliable preparations in which the anterior and posterior lobes are treated separately its use can only be experimental. From the standpoint of drugs, the indication is to keep up a good digestion, and the first indication is to assist in the digestion of the starches. Any one of the diastases will be found to be useful, preferably the *taka*-diastase.

Citrate of sodium and sodium formate have been recommended as palliative remedies on account of their well-known properties of preventing fatigue and of increasing the working energy of muscles. They are given in doses of 1.0 each (grams xv, t. i. d.).

Tonics in the form of iron, quinine, and strychnine and arsenic will assist in maintaining the general health.

Sudden attacks of dyspnea which threaten the life of the patient are to be combated by prolonged artificial respiration, for after a period of rest the respiratory muscles may recover sufficiently to again be able to perform their functions.

CHAPTER VI

THE TREATMENT OF THE EPILEPSIES

By J. F. MUNSON, M.D.

INTRODUCTION AND GENERAL CONSIDERATIONS

OUR present problem, the treatment of those conditions commonly grouped under the name "epilepsy," presents difficulties of solution which may be summed up as follows:

1. We are decidedly in the dark as to the pathology and the pathological physiology of all of these conditions and have only recently come to recognize the great diversity of the conditions involved.

2. Some of the things which are known about the disease and its treatment have been misunderstood, and, hence, wrongly used.

3. The characteristic features of the disease influence the life of the afflicted individual in every particular, and our therapeutic efforts cannot be confined to medication but must also extend to the social and economic relations of our patients.

Thus, obscurity, misunderstanding, and the wide range of problems involved, characterize this disease, which Spratling has aptly called the "strangest in human history."

The following cases will illustrate the treatment generally given to the epileptic, as far as can be judged from the stories told by patients coming into an institution:

The patient said that while he has always been well, he has experienced within the last few hours (or days) a peculiar attack, in which he lost consciousness. Those about him tell him that he cried out, as if in fright, that his eyes rolled, and that he fell heavily to the ground and shook violently for a minute or so, after which he quieted and lay in a deep sleep, breathing very deeply and noisily; he soon awoke to consciousness, with a headache and dazed feeling, bruised, and shaken, and with a sore tongue; he also found that he had wet himself. The patient is a young man of average appearance, showing one or two recent injuries, which he says were received in the attack. He had worked very hard the preceding day and had eaten a hearty meal just before the spell came on. No one in his family has had any similar trouble.

A second patient comes in with a severe burn of the hand, which he says he received while he was "not himself" after an epileptic attack. He has consulted many physicians (and quacks), and has been told by all that he suffered from epilepsy; the physicians mostly told him that the disease was hopeless—the quacks all promised speedy

cure through their treatment; he has spent much money in search of a cure for his condition. The medicines given him almost always had a salty taste and sometimes were "powerful" enough to stop the attacks, sometimes lessened them, sometimes had no effect at all, but almost always made him dull and slow, so that he rather preferred the attacks. He notices that he cannot remember and think as well as he used to, even when he is not taking the medicine. He comes both on account of the burn and for treatment for the epilepsy.

The first patient may be told that he has epilepsy, but there is almost an equal chance that he will be told that the attack is due to indigestion and probably will not occur again if proper discretion is used in working and eating. If the patient were a young child, the attack would be a "worm fit" or a "teething convulsion." In almost any event except the diagnosis of epilepsy, there will be practically no treatment ordered except some advice about eating; in case epilepsy is diagnosed, a prescription for some of the bromides will be given, and if it does not prevent the recurrence of attacks, the dose is increased without other change in the treatment. Exactly the same treatment will be given the chronic case; both patients get bromides and good advice and both go away from the office with renewed hope—for hope is characteristic of the epileptic; it is nature's way of offering compensation for the sufferings she permits; the epileptic always hopes—each attack is the last, each interval between attacks the beginning of a cure, each new prescription the panacea for his disease. Such seems to be the treatment in many cases, repeated over and over, until the chapter of their life is completed in an almshouse or other public institution.

Conversation with physicians in many cases confirms the impression derived from patients themselves as to treatment given. Many physicians regard epilepsy as a perfectly hopeless disease, which is on the whole uninteresting; bromides, the only remedy available, and one not likely to be successful. The case of epilepsy is considered a bore, to be gotten rid of as soon as possible, to give time for more hopeful and more interesting cases.

This deplorable attitude, unfortunately rather general, is not unjustified, because the vast amount of effort which has been expended in an attempt to cure or even ameliorate the diseases has been very largely unsuccessful. This is by no means surprising when we consider the difficulties of the problem, already mentioned in brief. But in spite of our ignorance of the diseases, and in spite of misunderstandings, we have methods of dealing with the diseases which make it by no means hopeless, which offer some prospect of betterment to almost every case—rarely complete cure; somewhat oftener "arrests" for varying periods; almost always improvement, either by the reduction of the number of attacks or by the lessening of their severity. No matter what the result may be in the end, every epileptic deserves a trial of these measures and it is with the view of pointing out methods of treatment, proved correct by experience and of urging their trial in all cases, that this article is prepared.

PLATE IX

Fig. 1



Diffuse Sclerosis of Cortex with Atrophy along the Rolandic and Sylvian Fissures (Epileptic).

Fig. 2



Gross Anatomical Lesions Associated with Epilepsy.
An asymmetrical internal hydrocephalus which also showed a small tumor at the lateral border of the cerebellum.

Definition and Etiology.—While the sphere of an article on treatment is necessarily limited, the writer wishes to present certain conceptions which he believes are not generally and correctly understood, and to this end must include a certain amount of material which is not strictly treatment, but upon which the latter in large measure depends.

The word epilepsy awakes in every mind a chain of unpleasant associations, more or less distinct, according to whether the person has ever seen an epileptic seizure. If he has, a picture is called up of a human being grovelling in the dust of the road, his humanity debased to the level of the struggling worm; it carries with it the impression of a mind temporarily blotted out, of a scarred and harassed body. In other words, our first and strongest idea of the disease is centred about the seizure, the major symptom of the disease.

It is only natural that definitions of epilepsy likewise make the disease centre around the paroxysm, sometimes with the mention of the tendency toward dementia; the ordinary definitions carefully recite all the characteristics of the seizure—the sudden onset, the changed or lost consciousness, the variable motor phenomena, and sometimes the etiology is touched upon in the mention of heredity as a prominent cause. Such a definition bears out the popular conception of the disease, namely, that the epileptic is such only at the time of his seizures and that in the intervals he is much like other people. As a corollary to this type of definition, since all seizures are alike in their essentials, there can be but one epilepsy.

This old idea of epilepsy as a disease entity is fortunately passing away in favor of the recognition of the disease as a *syndrome*, in which the seizure is the most striking of several symptoms—a symptom which is common to all forms of the syndrome because it finds its origin in the same kind of cells, which have one response to diverse stimuli. No one will doubt the general similarity of the attacks of the disease, or the fact of their origin in the central nervous system, so that the idea of the disease as a unit is not unjustified, or, in other words, the use of the singular “epilepsy” is not essentially incorrect. However, we must recall that the nervous system may be made to react in either of two ways—by changes within the cell, through which the liability of the cellular chemical mechanism is increased, or by the action of external forces, which temporarily overcome the equilibrium of the nerve cell. From such a conception of the origin of the disease it will be seen that our common manner of defining epilepsy as a disease of the nervous system is not always strictly correct; that we should rather describe it as a syndrome which may arise by action *within* or *on* the cells of the central nervous system, and that the similarity of reaction (symptomatology) in the two groups is due to the excitation of the same mechanism—or, as it may be expressed, the syndrome always arises by a reaction manifested *through* the nerve cells but is not always *of* them.

We have thus at once two varieties of epilepsy, differing in the

intrinsic or the extrinsic character of the source of the nerve-cell activity; these correspond to the groups commonly called idiopathic or genuine (intracellular) and symptomatic (extrinsic causes) epilepsies. It is to be recalled that clinically the group of idiopathic or genuine epilepsies is not a clear-cut group, but is rather a residual group, composed of cases in which we cannot recognize a sufficient extrinsic cause. A fairer name for the group would be "unclassified"—to term a disease idiopathic is only to disguise our ignorance. On the other hand, in the symptomatic epilepsies we are dealing with cases in which the syndrome of epilepsy arises from the action of some one well-defined cause, and accordingly our symptomatic group is really an etiological one, based on a group of agencies which, when applied to the central nervous system, act on the nerve cells and manifest their presence through them.

Such an etiological classification has one great objection, in that it seemingly specifies that any one case must arise from one definite cause, *i. e.*, that a case catalogued as "syphilitic epilepsy" is due solely to the action of the *Treponema pallidum* and its products. Our problem would be far simpler were this always so; there are undoubtedly such cases, but it is also true that there are many cases in which there is apparently a definite etiology, but which do not improve upon removal or treatment of the apparent cause. In some cases, especially those of longer duration, this failure to obtain improvement after treatment directed to the apparent etiological quantity may be due to the presence of chronic changes as the result of the disease—the changes themselves perpetuating the disease. On the other hand it seems more probable that the therapeutic failure is due in such cases to the fact that the syndrome is not the result of one definite factor, but rather to the sum of the activity of other causes, besides the apparent cause, *i. e.*, chief and contributing causes. This conception of the complex etiology of the epileptic syndromes is well expressed in the following form:

$(a+b+c+ \dots +m+n+p+ \dots +x+y+z)$ the brain = the syndrome.

This mode of expression, while mathematical in form, is not intended as a literal equation to epilepsy; it is to be interpreted as meaning that the sum of certain known causes of epilepsy (*a, b, c*) added to certain variable quantities (*m, n, p*) and to certain unknown agencies (*x, y, z*), all acting together on the brain, produce (=) the syndrome we call epilepsy.

Such an expression is in a way a definition of the epilepsies and has the following features of importance: It emphasizes the multiplicity of factors which may be active in any case; it shows, too, that since the component etiological factors may differ, there may be an almost infinite number of etiological complexes at the basis of epilepsy-syndromes, and hence points out that to some degree *each patient with epilepsy is peculiar to himself* and is both similar and different from all others; at the same time, by demonstrating the plural nature of the conditions underlying the syndrome it indicates a comprehensive

PLATE X



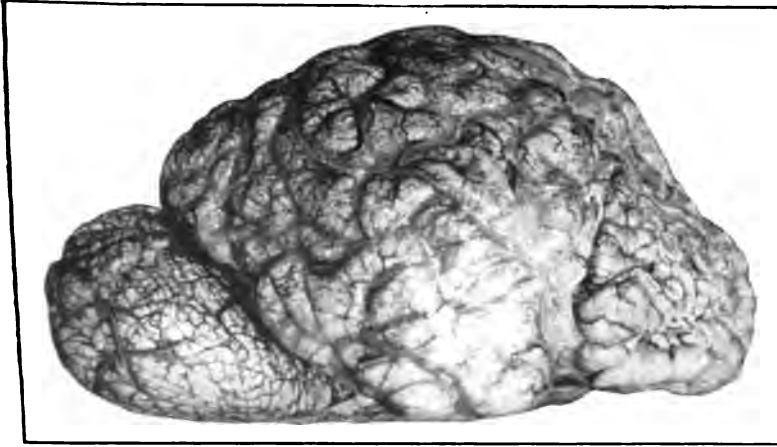
Gross Anatomical Lesions Associated with Epilepsy.

Asymmetry of the cerebral hemispheres and slight dilatation of the lateral ventricle. Both of these conditions are very common in autopsy material.

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PLATE XI

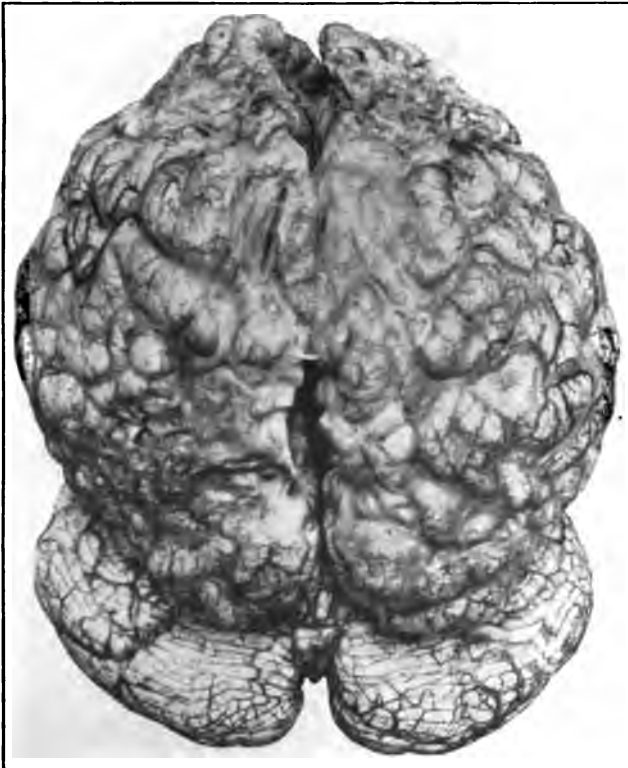
Fig. 1



Gross Anatomical Lesions Associated with Epilepsy.

Micrencephalon, with marked meningeal thickenings and localized regions of more extreme atrophy. Brain weight, 462 grams. Patient aged seven years.

Fig. 2



Gross Anatomical Lesions Associated with Epilepsy.

Micrencephalon, with marked meningeal thickenings and localized regions of more extreme atrophy.

mode of treatment, and explains why treatment along narrow conventional lines is so often unsuccessful. The treatment of *one* cause is not sufficient, since the variable or unknown etiological quantities may change to make the equation still equal to the syndrome of epilepsy.

The writer hopes that the conception of epilepsy as a syndrome (= symptom complex) of diverse origin has been made clear. The conception of epilepsy as one disease nullifies our efforts toward its elucidation and treatment by narrowing our conception of the pathology to nerve-cell changes and by laying insufficient stress on the causes which lead to their activity. The old idea of the disease attempts to unite diverse conditions on the basis of a common symptom—the newer while broadening the field for etiological study at the same time simplifies our task by dividing it into smaller fields of endeavor. It may be well, perhaps, to embody these views in a formal definition:

The epilepsies are a group of similar syndromes arising by action of the cells of the central nervous system through stimulation by various agents which may be either intrinsic to the nerve cells or extrinsic or from a summation of causes in both groups, characterized by seizures in which consciousness is altered or lost, with or without motor phenomena; characterized by mental changes and by certain traits of mind and character which exist independent of the seizure.

Classification.—Classifications are convenient scaffoldings on which to build up our knowledge of a disease process. But, unfortunately, in the epilepsies our efforts in this direction are far in the rear of those in the neighboring fields of psychiatry and feeble-mindedness; in both of these attempts at classification have led to a better understanding of the conditions studied. The old classification of the epilepsies, grand mal, petit mal, Jacksonian, and psychic, is of descriptive value alone, as it refers only to the type of seizure; to be complete, it should include another group made up of abortive, incomplete, and atypical attacks. The incomplete seizures is often misunderstood and often overlooked; partial grand mal attacks are often called petit mal and auras, which are certainly a definite component of seizures, and pass unnoted.¹

Turner,² of London, offers a classification in which the date of onset and the etiological factors are both considered:

¹ That there is a definite change associated with the aura in some cases is well shown in the case of J. C., No. 3214, on whom the writer was one day taking a blood-pressure reading. The mercury was at the top of the scale (300 mm.) and there was still pulsation at the wrist. Suddenly the patient remarked that he was going to have a seizure and asked for medicine. He showed no apparent sign of an impending attack, so the taking of readings was continued; finally a reading was obtained at 285 mm. and a short time afterward 245 mm. was twice obtained at short intervals; about this time he said that he was all right. This patient's blood pressure averages about 230 mm., and in this particular instance a subjective aura was associated with a high blood pressure, which returned to (his) normal after the aura passed.

² *Epilepsia*, vol. ii, p. 101.

Organic Epilepsies.—Focal brain disease and trauma.

Early Epilepsy.—Often marked by striking mental deterioration; includes some cases of infantile cerebral palsy, birth traumas, encephalitis, and hemorrhage.

Late Epilepsy.—May be due to cardio-renal-vascular disease; intoxications, among which alcohol is important (and syphilis and other infections can be best here included, except the focal manifestations of syphilis and tubercle); epilepsy associated with a psychosis; eclamptic conditions, developing into epilepsy.

Idiopathic Epilepsy.—Comprising the residue of all cases and having no ascertainable cause.

The Ligue Internationale contra l'Epilepsie offers the following terminology, which amounts to a classification: Varieties of epilepsy: Idiopathic, metabolic, late degenerative, traumatic, epilepsy related to syphilis, epilepsy arising from other, especially exogenous, causes.

It is evident from these diverse classifications that no grouping of the cases meets the requirements of all purposes. It is far more accurate today to speak only of

Symptomatic Epilepsies, with a qualifying adjective indicating the most apparent etiological factor, and

Unclassified, a group corresponding to the old idiopathic, but being a frank acknowledgment of ignorance.

While the various syndromes commonly called epilepsy differ from each other in etiology, their results are in many ways similar and admit of similar treatment and management; certain phases of the disease are common to almost all varieties of the syndrome and can be best discussed as considerations applicable to all epilepsies.

Prognosis.—The duration of the disease has almost always a direct influence on the result of treatment, and the longer the disease has existed the less the chance of cure or material improvement. The importance of the time element in the treatment of epilepsy is well illustrated in the following summary by Dr. Flood, of the Massachusetts Hospital for Epileptics:¹

Of all convulsive conditions, 50 per cent. are curable.

Of established epilepsies in young individuals, 20 per cent are curable.

Of old established cases, 5 per cent. recover.

Of cases with mental deterioration, 1 per cent. recover.

The importance of early treatment is here emphasized, and whether, as some claim, an epileptic habit is formed or whether chronic structural changes are produced in the brains of epileptics—changes which are themselves capable of continuing the disease—it can be seen that treatment must be begun before either of these processes are established if favorable results are to be produced. It is the general practitioner and family physician who first sees the cases, whose opportunity is greater than anyone else can possibly have, since he sees them at their onset, when no habit or chronic changes have been caused, and when favorable influences have their maximum influence.

¹ Boston Medical and Surgical Journal, vol. cli, p. 823.

PLATE XII



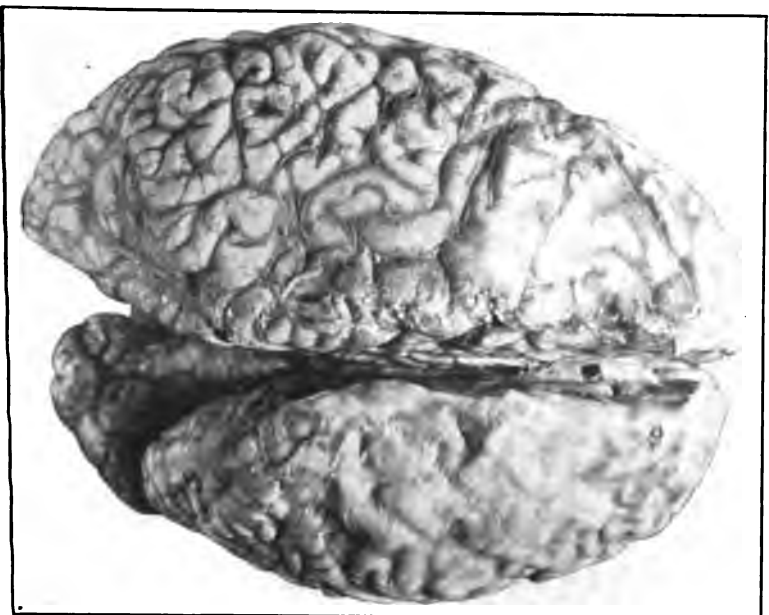
Gross Anatomical Lesions Associated with Epilepsy.

The brain of an epileptic of fairly advanced years, with general thickening and clouding of the arachnoid pia and well-marked Pacchionian granulations. Note the broad veins.

Fig. 1

PLATE XIII

Fig. 2



Gross Anatomical Lesions Associated with Epilepsy.

Cerebral asymmetry and ventricular dilatation following an early meningitis. The dura over the atrophic hemisphere was of the consistence of an egg-shell.

Gross Anatomical Lesions Associated with Epilepsy.

Cerebral asymmetry and ventricular dilatation following an early meningitis. The dura over the atrophic hemisphere was of the consistence of an egg-shell. Section of preceding brain.

Symptoms.—It must be noted, however, that the apparent onset of the disease is not always the real one; patients often recall on questioning that they have noticed incidents which are significant, in the light of the later developing seizures: Vertigos and headaches; a sore tongue in the morning, with great weariness; nightmares, etc., all of which may have been mild manifestations of epilepsy. The presence of such occurrences must be cautiously sought for in order that the duration of the disease and the prognosis may not be wrongly determined.

As indicated in Dr. Flood's table above quoted the mental status of the epileptic is an important factor in the prognosis of his disease. It is a common dictum among institutional people that there are practically no epileptics without some mental change. Progressive dementia is the most striking of these, and is the only one of the inter-paroxysmal phenomena which has received universal attention; it must be admitted, however, that there are epileptics who apparently do not deteriorate, but even for these the dictum will be found to hold, and a careful examination of them will reveal some mental abnormalities.

Apparent dementia must not be accepted at its apparent degree; in any case showing dementia there may be, beside the true dementia, from which recovery is impossible, the mental dulness and retardation of bromism, which frequently simulates or exaggerates a dementia. This bromine dementia gives surprising results under appropriate treatment—mainly elimination of the bromides—and patients who come for treatment saturated with the bromides, and consequently almost stuporous, gain wonderfully in mental (and physical) vigor. Dements also show a somewhat similar gain, though never to the same degree; the change of environment brought about by entering an institution awakes dormant faculties, which are, however, again submerged. The recovery from bromine dementia is permanent so long as the bromides are properly regulated.

Aside from either of these processes we find that epilepsy is very frequently associated with or engrafted on some degree of feeble-mindedness. Children who are feeble-minded may also be epileptic, and likewise when the mental defect is due to an arrest of development following an injury or inflammation the occurrence of epilepsy at once or later in life is not improbable. The diagnosis of the mental status, if it is to have value from a prognostic standpoint, must differentiate primary or acquired feeble-mindedness from that which is the result of epileptic dementia. Even children may dement, and it is only by careful consideration of the facts in the case that we can arrive at a conclusion. A few points of importance may be mentioned:

Has the patient always been of the present mental grade?

If at one time described as bright, average, or normal, what was the nature of the change? Was it sudden or slow?

Was the onset of the epilepsy associated at all with the change? Was there any infectious disease or accident at about the time of the change in the mental status?

What progress was made in school? how far did the patient go and why did he stop? Was learning easy or difficult?

If in business or at work, what degree of success was attained or why did he fail?

What is the mental character of the relatives?

Lastly, there is a series of very simple tests known as the Binet tests for mental age; these tests attempt to grade an individual by years of mental development, by comparing his ability to accomplish graded tasks with the capacity of normal children to do the same. The defect shown is often astonishing, and reveals to us the probable hopelessness of the case. The tests are not difficult to carry through, and require almost no apparatus beyond what can be improvised. As in all such work the personal equation of the examiner must be reduced to as small a factor as possible. (See Chapter on Feeble-mindedness, Vol. I.)

Many of the problems of the epileptic are affected by his mental status, are in fact practically the same problems encountered in dealing with the feeble-minded, so that a proper estimate of the mental status of our epileptics will have important bearing on the treatment we can give them.

In addition to the dementia and feeble-mindedness present in epileptics there are mental characteristics which are more or less peculiar to him. Aschaffenburg¹ lays special stress on the variations of the psychical condition (mood) which may occur independent of attacks. Grasset² characterizes them rather harshly, but nevertheless with much truth, and indirectly points out the variability of the psychic condition of the epileptic:

"On the one hand they are somber, taciturn, defiant, suspicious, always ready to fly into a passion, to hurt people, to become enraged, and to strike; on the other hand they are contrary, obsequious, obliging, wheedlesome, full of effusion and gentleness. In reality, epileptics are all or nearly all irritable, subject to attacks of sudden, violent, and ferocious transports of rage, during which they do not, as it were, belong to themselves. This irritability is the key-note of their character. Many have, in addition, vices and perverse instincts; many are greedy, violent. . . . They frequently have a tendency to a sickly piety or a sort of excessive religiousness, mixed with hypocrisy."

Jones³ of Toronto, in considering the mental characters of the chronic epileptic, lays stress on the dementia and describes it as a concentric narrowing of the field of interest, so that the patient becomes more and more confined mentally to matters in his own environment. This shows in the loss of memory, in which the recollection of personal matters is longest retained, while matter of general interest, such as history or school-learning in general, is first forgotten. The association reactions of epileptics are similar to those of imbeciles (naturally this

¹ Abstract from Journal of Mental Science, vol. lii, p. 789.

² Semi-insane and Semi-responsible, translated by Jelliffe, p. 171.

³ Transactions of National Association for Study of Epilepsy, vol. vii, p. 23.

PLATE XIV

Fig. 2



Gross Anatomical Lesions Associated with Epilepsy.
Internal hydrocephalus. Marked feeble-mindedness.

Fig. 1



Gross Anatomical Lesions Associated with Epilepsy.
Internal hydrocephalus. Feeble-mindedness.

would be true, since most epileptics are more or less defective), but differ from them in the greater complexity of mental processes shown. Jones places a very high estimate on the role played by sexual matters in the life of the epileptic, and traces many of the activities and characteristics to this foundation; with this we shall not concern ourselves, except to note that the epileptic is apt to be decidedly erotic, and that this must be borne in mind in considering measures for the prevention of the increase of the disease through diseased progeny. The egocentric make-up of the epileptic is well illustrated in the extreme attention which he pays to the state of his health and to his minor bodily functions; some epileptics are always ailing, always having some symptom to report, of which the commonest is constipation—a complaint which, however, is usually a valid one in epileptics. A dose of medicine will make some epileptics extremely happy.

The epileptic is full of contradictions and contrasts. Aschaffenburg calls this a periodic variation of psychic condition. Grasset speaks of it as irritability. It appears to the writer that we might also characterize the mental poise of the epileptic as extremely unstable. The instability, irritability, and variation of his mood make him difficult to deal with; these states are seen not alone in connection with the seizure, but also without apparent cause; therefore the handling of epileptics requires the greatest tact and discernment in order to determine what is really sullenness or what is a pathological state of mind. This does not mean that the epileptic should be humored in his ways; it means, however, that there are periods when he is best let alone and other times when he should be required to submit to discipline. Rules cannot be laid down for this—tact and understanding must guide one in the individual case.

A trait which appears uppermost among institutional epileptics is laziness. Institutional epileptics, as a class, prefer to avoid work, and apparently this preference is also seen among epileptics outside of institutions. It may be a tendency related to the disease, but is more probably based on the undisciplined life which most epileptics live outside of institutions. As soon as their seizures are known it is almost hopeless for them to obtain employment, and what they can obtain is usually haphazard and at irregular intervals. Combined with the lack of initiative of the enfeebled or demented mental condition the result appears as laziness.

In most epileptics there are periods varying greatly in duration and character in which the epileptic is rightly to be considered irresponsible even though at other times they approximate a normal mental state. With some epileptics these diseased states are continuous, and the patient may rightly be classed as having an epileptic psychosis. Mostly, however, these abnormal mental states last only a few hours or days; it would be an injustice to such cases to keep them confined continuously, and yet it is equally impossible to allow them to be uncontrolled at such psychotic periods on account of the injury they may do themselves or others.

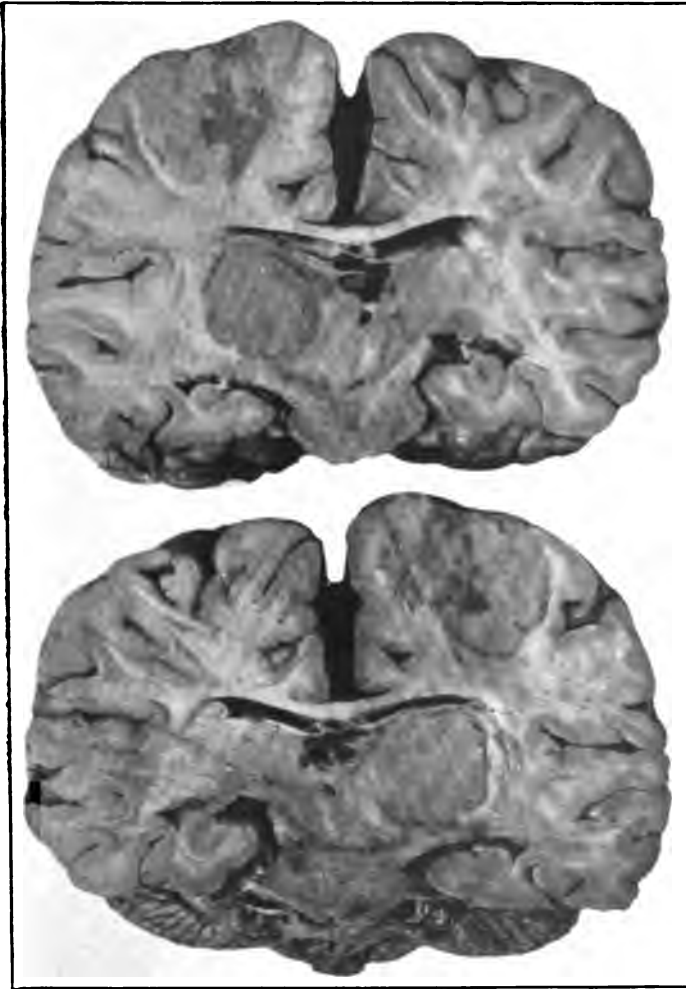
The character of the periods varies greatly; in institution vernacular they are classed as "mental confusion" or "mental disturbance," the latter being confusion plus more or less excitement. Confusion is the simplest of these conditions—the individual is disorientated to a greater or less extent and is sometimes almost completely inaccessible. On the other hand exaltation states are particularly common—orientation for time and place being ordinarily fairly well retained, and the basis of the happy frame of mind is most commonly the suddenly appearing belief that he [the patient] is cured or that a method of cure has been pointed out to him, sometimes by Divine revelation. Sexual ideas occasionally enter into these mental states, but ideas of cure and of religion are most common.

Maniacal episodes are not uncommon with some patients and present a source of danger to those about. It is not the sphere of this article to describe these states but rather to point out their existence, in order that they may be expected and guarded against. A particularly serious character of all these mental states is the tendency of the patient to wander. Patients will slip away by mere unintention; they accidentally pass beyond the observation of their attendants and do not know how to get back—probably in many cases do not even realize that they are away. Sometimes in these wandering states the patient jumps out of windows or off porches, or even climbs on to the roof; or travels toward nowhere in particular at a marvellously rapid pace—very often unclothed and in the midst of winter. Similar happenings occur in the postepileptic states, and therefore epileptics should be constantly under observation.

These after-seizures and, more rarely, preconvulsive mental states are extremely serious if wrongly handled. Much depends on the individual case; some do not seem to have a tendency to violence and can be controlled with some ease; in other cases the slightest attempt to regulate the movements or actions of the patient will provoke violent resistance. As will be repeated many times a knowledge of the individual is of the greatest importance in these cases.

Liability to Injury.—Injuries dependent on the unconsciousness and helplessness due to seizure and its associated states are very common in the epilepsies. The isolated instances of this sort which come to the notice of a man in general practice do not give an adequate idea of the great dangers to which the epileptic is exposed through his attacks. Cuts and bruises are common, but, unfortunately, serious injuries and even death are not uncommon results of the seizures. It is part of our duty in the outlining of the general characters of the epileptic to emphasize this point. His whole life must be arranged in order that he may not be needlessly exposed to these dangers—he must not work or climb to heights from which he might fall; he must keep away from fires and hot stoves lest he fall and burn himself; he must keep out of the way of moving machinery; he should be careful in crossing busy streets; he should not bathe nor stand in water lest he drown. At all times he should be under observation. In institutions, where

PLATE XV



Gross Anatomical Lesions Associated with Epilepsy.

Tubercles in corpus striatum and in cortex.

the expense of supervision would be great otherwise, great reliance is placed on the care which one epileptic will always give another. The chance that two epileptics alone together will each have a seizure at the same time is remote, and this pairing will often enable paid supervision to be dispensed with to a great extent. There are a few instances in which the aura is so distinct and prolonged that the patient prepares for the attack, and with such patients greater liberty may be permitted.

However, experience shows that the most serious danger which the epileptic has to face is death in bed. Here, if anywhere, it would seem that he should be safe, but experience shows that death during the night is the greatest accidental danger to which he is exposed. There seem to be three kinds of these deaths: (1) Where the patient rolls on his face and smothers while unconscious; (2) where the patient succumbs to a rapidly developed pulmonary edema; in this the patient practically drowns in his own transudate; (3) cases in which there is nothing to explain the death except a dilated right heart; in these cases paralysis of the respiratory centres has been suggested. The proportion of accidental deaths among epileptics from causes directly related to a seizure was about one-sixth the entire number of deaths in a series of nearly 600 deaths.¹ These things emphasize one of the cardinal requisites of any plan of treatment or mode of life designed for epileptics that they must always be under the closest observation.

Social and Economic Relations of the Disease.—The seizures and the mental changes of the disease awake, even in the most sympathetic, a certain amount of repulsion; in the ignorant this amounts to an absolute refusal to work with or associate with the unfortunate victim of epilepsy. The result is the social and economic isolation of the epileptic. Where the patient has ample financial resources this is not serious, as an ample expenditure will assure proper care and support. With those of small means, where each member of the family must contribute toward the family income, this isolation is serious and represents a different sort of situation. The epileptic is unable to obtain work (1) because of the objections of his coworkers, and (2) because his disease makes him an uncertain and undependable worker. Many epileptics tell of discharge from place after place as soon as the fact of their disease became apparent, and others relate a gradual lowering in the grade of work they could do as the disease progressed. On the other hand we occasionally find that the disease is for some time successfully concealed and that the patient is able to continue his work in an ordinary manner; these cases are sometimes a menace to the general

¹ The diagnosis of seizure death must rest usually on negative evidence, as there is nothing absolutely characteristic of the condition. General congestion, especially of the splanchnic area and lungs, with edema of the latter, dilated right auricle and ventricle and hydrops of the subarachnoid space are the ordinary findings. The dilated heart is most constant, after which would come the general congestion of the splanchnic area and of the lungs, with edema of the latter. The situation of the body when found must be carefully considered; the position of the fingers (closed over thumb in palm), petechial hemorrhages in the skin or conjunctivæ, urination or defecation, evidence of mucus or fluid from the mouth are of value. See Medical Record, Death in Epilepsy, January 8, 1910.

public, because their seizure *might* occur during working hours. A most striking example of this kind was a patient who had apparently retained the position of switchman on a railroad, after he knew himself to be an epileptic, and had thus endangered many lives. Where the seizures are nocturnal, or very mild petit mal, they may be successfully concealed.

The financial stress which results from the exclusion of the epileptic from regular work—day labor and “odd jobs” are usually the extent of the employment he can obtain—affects not only the support which he can give a family but also the treatment which he can receive. Ample finances greatly lighten the task of treatment, and on the other hand, poverty or even moderate circumstance renders the task of establishing a proper home treatment of the disease one of extreme difficulty, if not of impossibility.

The treatment of any of the epilepsies must take into consideration these factors which affect all epilepsies alike; the treatment must be fitted to the individual case and can only be planned in the light of a complete medical and social examination of the epileptic and his environment.

TREATMENT

General Medical Treatment.—As has been said, our knowledge of the etiology of epilepsy is limited and the group of cases which must be called “unclassified” in an etiological classification is far larger than that which can be called symptomatic. In the latter group, we may attempt symptomatic treatment, but in the former we have only at our disposal general measures which have empirically or otherwise, proved of some benefit in the disease. Likewise, in the symptomatic cases, general measures must be employed to care for contributing causes so that the general medical treatment of the disease is of first importance.

As has been said, our treatment is very largely empirical. Attention has mostly been paid to the convulsion, and attempts have been made, in one way or another, to so reduce the irritability of the cortex as to stop the disease; and except by those who regard epilepsy as an auto-intoxication, little other treatment is used. The colony care of the epileptic is the outgrowth of a social need and its extension due the recognition of its therapeutic merits by medical men. We have no specific therapy—which sedation is not—but we are in possession of methods which rightly handled will often yield favorable results.

Bromides.—The treatment of epilepsy, where epilepsy was regarded as composed of the convulsion alone, rested on the properties of various drugs to inhibit the activities of the nerve cells of the cerebral cortex. Bromides have proved the best of these sedatives and have been so extensively used that epilepsy and the bromides are almost synonymous. As indicated earlier in this article, the first treatment usually given an epileptic is a course of the bromides, given sometimes to cure, but

usually simply to stop or moderate the attacks. No greater injustice than this could be done the patient. There are some who believe that the bromides are curative in certain mild cases, in small doses over long periods—one distinguished English neurologist so stated his belief to me. It is now generally accepted that the bromides are not curative but only palliative, and are to be given only as such in chronic cases, or in recent cases, as a means of controlling the seizures while other treatment is being carried out.

The old methods of bromide administration resulted in doses of as high as an ounce or an ounce and a half in a day of the potassium salt; naturally in most cases the seizures were stopped, but the result for the patient was worse than a moderate number of seizures—he became dull, stupid, apathetic, suffered from digestive disturbances and from an acne which was exceedingly annoying, especially as the acne often became infected, leading to furunculosis. Improvement or arrest of the seizures from bromide administration was not permanent and the patient relapsed into his former or a worse state as soon as the bromide was diminished or withdrawn. Patients themselves comment on the effect of bromides and sometimes express a preference for a few seizures rather than somnolence from bromides.

The true sphere of the bromides, as we see it today, is to effect a balance between the number of seizures and the condition of the patient, and should only be used where other measures have failed. This is a radical change from the idea of a few years ago, and one which has resulted in increased comfort for the epileptic. Only a minority of patients require bromides as a routine treatment, if they are properly looked after in other ways. These cases, however, require a small amount of bromides to prevent a too great number of attacks, which if unrestrained might be dangerous. A stock formula of use is the following:

	Gm.
R—Potassium bromide	8.0
Sodium bromide	8.0
Water to	100.0

Of this a teaspoonful represents about 10 grains of the mixed bromides and the dose can be thus arranged to suit the needs of the patient. *The effort should always be to give no bromide or as little as is consistent with safety to the patient.*

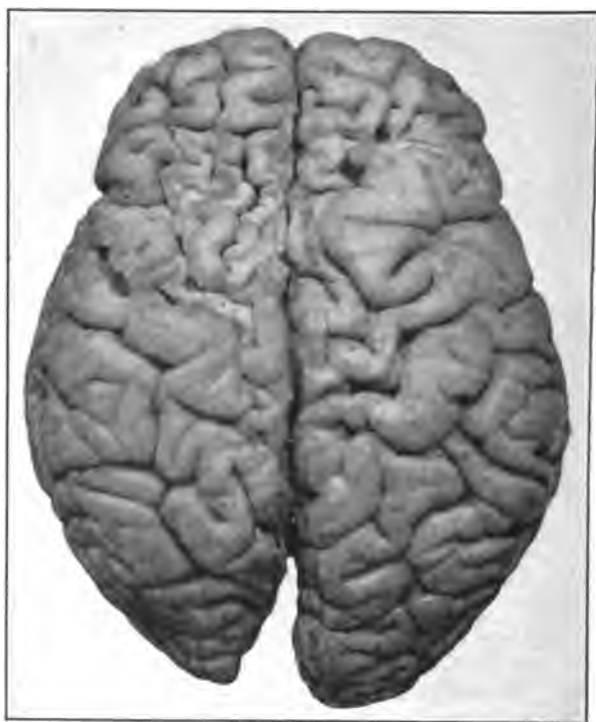
Where bromide is to be given regularly, various methods may be used to administer it. It may be used instead of salt in bread, and rolls baked of the proper size for one dose. An artificial mineral water was used by Collier¹ with success; the bromide was combined with magnesium sulphate and sometimes with iron to make a concentrated watery solution, which was diluted as required. Twelve parts of the mixed bromides of sodium, potassium, ammonium, and calcium are dissolved with 8 parts of magnesium sulphate to make 128 parts of concentrated

¹ Transactions of National Association for Study of Epilepsy, vol. iii, p. 39.

solution. Of this 2 ounces diluted to a quart will give about 11 grains bromide in a half pint of water (one ordinary tableglassful) and about $7\frac{1}{2}$ grains magnesium sulphate.

Several compounds in which bromine is substituted into an organic radical have been placed on the market, with the object of preventing the gastric disturbances which sometimes follow the administration of the bromides as metallic salts. The substitution of bromine into a vegetable oil yields a preparation which has some advantages, but, on the whole, these modifications have no advantage over the metallic bromides when the latter are properly used.

FIG. 15



Gross anatomical lesions associated with epilepsy. Localized atrophic changes of unknown origin, but probably traumatic (birth injury) or the result of an old inflammatory process.

Preference is expressed by some for a particular metallic bromide or for a particular combination; it seems probable that these are variations of slight importance, that one is as good as another.

In discussing bromine therapy, reference must be made to the numerous nostrums which are sold as "treatments" or "cures" for epilepsy. It is entirely unnecessary to call attention to the pernicious character of such medication. Mostly these nostrums contain bromides in large amounts, even though statement is made to the contrary, and

their success in arresting the seizures simply depends on the enormous dosage of bromides which the patient is directed to take.

It used to be that the face of every epileptic was covered with acne, from the heavy bromide medication he was receiving. Fortunately, this is not so true at present, but nevertheless, many patients come for treatment who are thoroughly saturated with the bromides and who show the symptoms of bromism—mental dulness and acne. The clearing up of such cases is something which must be done with great care, as a sudden reduction in the amount of sedative in the body will not infrequently result in an excessive number of attacks—even serial seizures or status epilepticus; if the drug is withdrawn suddenly, every precaution must be taken against an emergency. The clearing out of the bromides should be done fairly slowly; an attempt should be made to determine the amount of bromides which have been taken and to start from this point and work the dosage downward with only a moderate rapidity.

In some cases, a condition of delirium results from the bromine intoxication; this is extremely exhausting, and it may seem advisable to produce rapid elimination of the bromides. (See Chapter XVI.)

The urine is the main vehicle in which the bromides are excreted, but at the same time the bowels and skin are valuable aids. Diuretics and continuous normal saline per rectum will probably do the work best. The salt contained in the saline solution will have an almost specific action in forcing the elimination of bromides, an increase of the sodium chloride concentration tending to force sodium bromide out of the body. Doses of common salt may even be given for this purpose, but it must be borne in mind that any rapid excretion of the bromides may be *exceedingly dangerous* to the patient and should only be undertaken in cases of bromide delirium. The physician must be prepared to control with chloral or other sedative the seizures which may result.

Reduction of the bromides is of course a necessary part of the treatment of bromide acne, but the simultaneous administration of arsenic—Fowler's solution—seems to restrict its occurrence. Baths are of use in the treatment of the bromide eruption.

A form of cure which had many advocates at one time was the opium-bromide treatment of Flechsig. This consists essentially of a course of treatment with opium in ascending doses, extending over a period of six weeks, and reaching 15 grains a day; the opium is then withdrawn and bromides in 120- to 130-grain doses daily is substituted over a period of two months, after which the dose is gradually reduced to 30 grains a day. It was recommended only for chronic cases where the bromides alone had failed and was to be carried out under the closest observation. The consensus of opinion seems to be that the treatment is not successful.

Other Sedatives.—Practically no other sedative than bromide can be used in the routine treatment of epilepsy. A long list of remedies might be given—*adonis vernalis*, *solanum carolinense*, and *simulo* are among the vegetable remedies recommended as a routine, but they are

of use, if at all, only in rare cases. On the other hand the sedatives of the synthetic group are at times indispensable, not for routine, but for emergency use. In the conditions of rapidly recurrent seizures and in mental disturbance, chloroform, chloral, paraldehyde, amylene hydrate, etc., have their usefulness. They are habit producers or otherwise unsuitable for routine use.

Borax has been extensively used as a specific against epilepsy, and as with all remedies, favorable results have been reported. It has practically no advocates today; its effects on the digestive tract and skin are such that it is not a desirable treatment. The work of Dr. Wiley on normal people with small doses repeated over a long time is additional argument against it. It has been given in as high doses as 120 grains per day.

In cases where there is a prolonged prodromal period and in which the seizures are comparatively infrequent, sedation should never be continued through the interval but only administered when the attack threatens. Similarly, when a patient has seizures with some regularity at a given time of day, as some do, a somewhat larger dose of sedative one to four hours before the ordinary time of the seizure will probably be more beneficial than a much larger amount distributed through the day.

The fundamental principle of routine sedation in the treatment of epilepsy is wrong. It is an attempt to prevent the nerve cell from reacting to a stimulus or condition and is not an attempt to remove this stimulus or condition. Sedatives can never be more than palliative—true, in the emergencies of the disease they are life-saving but never curative; no treatment which depends on putting the brain to sleep in whatever degree can be; the successful treatment will be based on the etiology of the condition and will strike at the cause and not at the mechanism. Stopping an engine by means of a brake without shutting off the steam is exactly analogous to trying to stop epilepsy by sedatives.

As we have already stated, the fundamental characteristic of the epileptic as we see him is an unstable nervous system—a nervous system whose equilibrium is upset with great ease by various causes. Sedatives serve as props to the tottering structure and support it only long as they are continued—what we have to do is to strengthen the nervous structure of the epileptic so that it can resist the causal agents of epilepsy by itself, so that its equilibrium is more stable and less ready to respond with a seizure to certain stimuli. The maintenance of equilibrium by means of sedatives is an undesirable method and one which brings no permanent results.

Hygienic Treatment.—As a substitute for the bromides and as a real curative agency we rely on a multiplicity of methods which may be summed up in two words, "good hygiene." Good hygiene in this sense means something more than obedience to the laws of health; it means good food, good sanitation, proper exercise—every means which places the body of the patient in the best physical condition; more than

this, it contains the idea of a discipline which will inculcate self-control. Personally, the writer believes that obedience to discipline and the resulting gain in self-control is no small part of the treatment which is summed up in good hygiene.

It is a difficult matter to present in any definite way the rules of life which should guide the epileptic. In fact, the individualization of the treatment of the epilepsies is so important that hard and fast rules cannot be laid down and only the general principles can be indicated. What might be good for one case will fail with another. It is in carrying out the hygienic treatment of the disease that the practitioner is most helpless. The difficulties in enforcing any such treatment at home are very great; the busy mother finds it hard to spare time to oversee her epileptic child; the ignorant fail to apprehend the wisdom of the measures advised and deliberately violate them; while many, through their great love for the afflicted one, cannot deny them anything they desire and indulge all their wishes. If therapeutic results are to be obtained from hygienic measures, either the home must be of such a character that perfect coöperation may be obtained or else the patient must be removed to a suitable hospital or institution.

To everyone who has to deal with large numbers of epileptics there comes the impression that the gastro-intestinal tract has a very great share in the causation of the individual attacks and emergencies of the disease. This does not mean adherence to a hard and fast auto-intoxication theory of the disease, but it does imply a belief that absorption from the gastro-intestinal tract is one of the important variable quantities which enter into the summation of causes which lead to the seizure.

Diet.—One of the chief characteristics of the epileptic, closely related in all probability to his unrestrained will, is the tendency to overeat, to gourmandize. In the great question of diet, *limitation of the amount of food taken* is of far greater importance than selection of the articles to be consumed. Naturally, the minimum amounts of the various food principles are quite definitely fixed by physiological demands; there seems to be an accepted belief that nitrogenous foods should be kept at or near the physiological minimum, but that beyond this there is not any accepted basic principle for the selection of the epileptic's diet.

The choice of the individual articles of food which are to be permitted depends largely on the individual experience of the patient. Many patients are aware, from experience, that certain things are bad for them and, on the other hand, feel that others can be taken with impunity. This being the case, the epileptic should not be tempted to indulge in the undesirable articles, but need not be denied those foods which do not cause noticeable difficulty—provided always that the quantity is carefully controlled.

Candy, sweets, and rich pastry are common sources of difficulty. These represent special dangers when patients are living at home,

especially if in a town, and after visits from friends to patients in a hospital or institution, when dainties are smuggled into the patient through mistaken affection.

Fruits are ordinarily desirable; no objection is apparent to the citrous fruits, but to some of the heavier and more pulpy varieties, for example, bananas and apples, at least when unripe, there seems to be reason for objection. In the case of unripe apples especially, and sometimes from improperly prepared dried apples, trouble is almost bound to come; and in the summer, while the fruit is on the trees, great vigilance has to be used to prevent an indulgence which may cause a dangerous run of seizures.

Alcohol occupies a prominent place in the list of forbidden substances; there can be no question but that it is a more dangerous poison for the epileptic than it is for normal people, and some go so far as to describe its action on the epileptic as being almost specific. In some cases this is true, and an alcoholic indulgence is inevitably followed by a seizure; in some there are seizures at no other times. Without denominating it a specific poison for the epileptic, its absolute restriction is to be desired in all cases. On the other hand, in the conditions in which it is used therapeutically, as in pneumonias, it seems to have no ill effect on the epileptic as such. It should be cautiously used, however, where cases are known to be susceptible.

Alcohol may also act as a sedative in epilepsy, though its use is not advised. A patient recently under observation had been encouraged (at another institution) to use alcohol to dull the anxious state which preceded his seizures by some hours. This patient controlled himself here very well for some months, but finally during an anxious state slipped away to the neighboring village and became intoxicated. The interesting thing about the incident was that he did not have a seizure so far as is known and that he cleared up much more rapidly and better than after bromides. Bearing in mind the habit-producing power of this drug it might be a valuable sedative in some cases. The writer is not to be understood as recommending its use for such purposes.

Of individual articles of food, articles prepared by *frying* are generally undesirable on account of the difficulty of digestion. Some patients recognize their inability to eat greasy foods, and it is in general wisest to prohibit such dishes. Certain heavy foods, such as pork, cabbage, etc., have been generally condemned, but it is our experience that unless the individual patient experiences difficulty from them they do no harm when occasionally included in the dietary in limited amounts. A meal containing pork or one containing a little cabbage will do no harm occasionally; it is probably best that the pork should be roasted very thoroughly and that the cabbage should be finely chopped. As regards cabbage and similar bulky foods, they have a distinct usefulness in giving bulk to the feces and thus promoting peristalsis. All varieties of pastry are not to be condemned; simple cakes and cookies and puddings made from rice or tapioca or similar material are entirely suitable and may be permitted; the kinds commonly characterized

as rich are to be forbidden. Fruits as specified above are desirable and should be taken without too much sweetening.

The whole secret of diet in epilepsy lies in moderation; this must be the key-note, and if the patient cannot control himself in the matter of eating the food must be portioned out to him in quantity sufficient to maintain body weight at a proper level. Some recommend under-nutrition (associated with thorough elimination) for a time, and this may have a valuable role in clearing out from the body the products of intestinal putrefaction. There is no one way which is best for all epileptics—what is good for one will fail with another. This is well shown by tests of some special diet with groups of epileptics, as for example milk, an exclusively vegetable diet, etc.; individual cases are benefited while others are unaffected or are made worse. It is entirely a matter of moderation and of the individual case.

Closely allied to the choice and amount of the diet is the matter of its digestion and the elimination of waste products. The proper selection of diet naturally has a great influence on the processes in the lower part of the digestive tract, but attention to this alone is insufficient. The condition of the digestive tract in all epileptics seems to have some influence on the attacks, but we cannot say definitely whether this is causal or merely coincident. We know, however, that in many epileptics constipation precedes the attack; we know that indiscretions of diet bring on attacks; and lastly, we know that clearing out the bowels during prodromas will often abort a seizure. Apparently something in or coming from the digestive tract acts as an excitant on the nervous system and brings on the attack. In fact, the writer does not hesitate to say that elimination, especially from the bowels, is one of if not the most effective weapon we have in the treatment of all epileptic conditions.

Treatment of Constipation.—Constipation is the constant complaint of the epileptic. In part, this is simply a manifestation of the ego-centric character of the epileptic makeup—it serves simply as an excuse for attracting attention to himself—but in large part it is a real condition and requires attention. The clearly marked condition, where there is cessation of fecal discharges for a time, is always recognized and treated as soon as brought to the notice of the physician. The more insidious variety, which might be called a relative constipation, depends on a slowing of peristalsis, with the result that the intestinal contents are retained for a longer time than normal. Someone has described this as having the stools a day late. There is thus increased opportunity for absorption and bacterial change. So far we have no experimental proof that there is such absorption, but the clinical association of constipation and increased attacks certainly indicates some sort of relationship.

The simple diet recommended for the epileptic and the regular muscular work which should also form a part of the treatment will be valuable aids in promoting peristalsis; there is usually, however, a long standing habit of constipation to overcome, and both training

and direct treatment are usually required. We have to educate the intestinal tract to a regular and more active habit of peristalsis, the chief of which is the cultivation of a regular habit of going to stool at a definite time of day. Nevertheless, even though a daily movement be reported, it is profitable to occasionally make sure that the passages are sufficient and that material is not being accumulated.

The great difficulty in making this sort of treatment effective is to make the physician and patient understand that it must be extremely thorough and prolonged and to get the patient to cooperate in a treatment which is often inconvenient. For instance, the physicians whom the writer has observed seem mostly to regard a dose of calomel, followed by a Seidlitz powder, or a single purgative pill, or an enema, as sufficient treatment for constipation. The writer is indebted to one of his colleagues, Dr. Collier, for the repeated demonstration that such treatment in epileptics is insufficient and that incomplete purgation may even promote trouble by stimulating activity in the large intestine through the incomplete removal of old material and the introduction of some new.

The necessity of thoroughness in clearing out the lower digestive tract cannot be overestimated. The individual should be placed in bed on very restricted diet or on no diet at all and purgation and enemas should be continued for two or three days. It is surprising in some cases the amount of fecal matter which is obtained late in such a course of treatment; the rectum and sigmoid apparently are easily emptied and probably well up into the transverse colon, but in the cecum and ascending colon there is likely to be retention of fecal matter, which will act as an irritant unless removed. This is sometimes shown at autopsy in cases of fatal status in which in spite of active elimination there is still retention of material high up in the large intestine. Failure will inevitably result from this method unless thoroughness is used in carrying out the treatment; and half measures are entirely unsatisfactory.

Where some purgative is required for daily use cascara is the choice in most cases, though in others Epsom salt is more successful. Occasionally a daily enema is more desirable.

Having in mind the importance of the lower digestive tract where bacterial processes play an important part, both in the desirable and undesirable processes, it is but natural that oral and dental hygiene should be important. Carious teeth and accumulations of tartar and of mucus about the teeth and throat serve as reservoirs of infection, from which a continuous stream of bacteria is sent out to perpetuate the conditions in the lower parts of the alimentary canal.

In beginning the treatment of a case of epilepsy the services of a dentist should be called on quite as much as the services of an ophthalmologist or other specialist. He should do a complete piece of work, fill all cavities, extract all hopeless stumps, and furnish artificial denture if required. The patient should be reexamined and the teeth cleaned at frequent intervals. Besides this work the patient should

be furnished with a good tooth-brush and with a pleasant mouth wash—the pharmacopœial liquor antisepticus alkalinus and acidus are very suitable and comparatively inexpensive. It is not sufficient that he be given these materials—someone must see that he uses them regularly three or four times a day. The essential part of the process is the mechanical cleansing with the brush, and the particular mouth-wash used is more or less immaterial.

The *restriction of salt* in the diet of epileptics has been very highly recommended. Very much is claimed of this procedure, but its probable value does not extend beyond the enhanced effect of small amounts of bromides through the lowered sodium chloride concentration, the reverse of the mass action already mentioned in connection with the rapid elimination of bromides by the giving of common salt, in which the excess of salt tends to force the bromides out of the system. Possibly in cases where a cardio-renal-vascular condition complicates or causes the epilepsy the deprivation of salt may be especially efficient, but on the whole it is to be valued only as a means of getting large effects from small doses of bromides.

Drugs Used in Treatment of Epilepsy.—A number of other drugs are at times used in epilepsy. *Morphine* is, on the whole, contraindicated and need not be given in the treatment of the disease. *Digitalis* has been recommended in the treatment of epilepsy as a means of improving the cerebral circulation, in the belief that a cerebral anemia was the underlying cause of the attacks. It does not seem probable that this result will be attained, except in cases where the heart shows evidence of muscular disease. In epileptiform attacks accompanying auriculo-ventricular dissociation the immediate cause is asystole, and digitalis will probably improve the condition. In the past there were epilepsies which were termed heart or cardiac epilepsies, but except in the case of the Adams-Stokes syndrome we cannot trace the connection of heart and epileptic seizures. An accompanying heart condition is to be treated as such, irrespective of the epilepsy.

Upon the theory that minute hemorrhages in the cortex determine the individual attack, and that these hemorrhages are due to a low coagulation time of the blood and also upon the idea that it has possibly a relation to ductless gland activity (as in tetany) and to the production of tissue edemas, calcium has been recommended in the treatment of epilepsy. There seems some evidence to show that in many epileptics the coagulation time of the blood is slowed as compared with the normal. The calcium is given as the chloride, as the lactate or lactophosphate, but its use has not led in general to any startling results. It is like many other things, a remedy which may be tried and which may succeed in occasional cases.

Thyroid extract, parathyroid, and other organ preparations have been recommended. The future may possibly reveal to us the role played by the ductless glands in epilepsy. There are cases which improve under administration of the organic preparations, but we do not at present know the kind of cases in which this occurs, or the

mechanism, well enough to lay down rules for the use of these substances in the treatment of forms of epilepsy.

Valerian is sometimes used in cases in which there is apparently an hysterical element.

Some years ago a considerable amount of work was done with the serum¹ of epileptics in an attempt to demonstrate a poison and produce antitoxic substances. Ceni reported the first of this work and presented results which appeared hopeful. The method was not successful in the hands of other observers, with some exceptions, and the work has passed into disfavor.

The use of crotalin or rattlesnake venom has been advocated by Spangler,² who reports very favorable results. The writer cannot perceive any theoretical grounds for such treatment, and the favorable results obtained are probably only those obtained for a time from any change of treatment and improved conditions.

Occupation.—A part of the medical treatment of the disease in all its forms is a suitable occupation. Unfortunately, the epileptic soon finds that the malady from which he suffers is both reason and excuse for his not working, and in some cases it is even suggested that work is inadvisable. The writer recalls an epileptic who said that his doctor (at home) had told him that work was bad for his disease—never was physician's advice more willingly followed. Work is advisable for all epileptics to the extent of their ability, both for medical and economic reasons.

Productive work, on a par with that produced by average normal people, is out of the question for the epileptic; he will rarely be able to support himself as he could before he became epileptic. It must be realized that he is under serious handicap—that the periods of idleness enforced by the attacks and the mental deterioration of the disease will steadily undermine his productive ability. But no matter how small his efficiency, he should attain as near a maximum production as is possible for him. From an economical standpoint he will be contributing something toward his support so that he does not represent an entire loss to the community or become so great a burden on his family. Medically, the value of work lies in the fact that *regular* work is an excellent form of discipline, and discipline of whatever form is valuable in overcoming the fundamental instability of the nervous system of the epileptic. The ability to stick to a given line of work means a gain in self-control and represents an inhibition which spreads to all the cerebral activities. The downward tendency of the disease is sufficient without aiding it by allowing the epileptic to make of himself a hopeless wreck, a derelict, an object of charity. Work makes the epileptic proud and gives him stability.

The exact kind of work is immaterial so long as it does not place the epileptic in positions where his seizures expose him to danger.

¹ Literature listed, *Journal of Nervous and Mental Disease*, May, 1907.

² *New York Medical Journal*, xcii, 462.

Only in rare cases, where the character of the aura is pronounced and well-recognized, should any risk be assumed, and then only under observation. It must be remembered that epilepsy is a dangerous disease. Outdoor and manual work are to be preferred over indoor or mental occupations; experience shows that epileptics do not do well at occupations requiring much mental effort. Farm-work, because of the happy combination of fresh air and work and because efficient oversight can easily supply the defects of the patient's mentality, is on the whole the best occupation which can be provided. Other manual pursuits which may have been learned before the onset of the epilepsy may be continued, with proper safeguards if there be danger of accident.

Keeping the epileptic at work will in many cases fall on the physician; the combination of the horror and dislike of the epileptic on account of his seizures with the natural feeling that an invalid should not work, will make the providing of an occupation for the epileptic a task of some difficulty. If the physician desires to really benefit the case he must see that a regular occupation is provided and followed. It may require a good deal of ingenuity to provide work for these people; in England, and also lately in Philadelphia, there are societies whose business it is to provide occupation for the epileptic. Work for the epileptic is indispensable.

Regular work is associated with a regular routine of life. The epileptic should rise, eat, and retire at regular hours; the meal-times should be fixed; the hours for work should be clearly defined and there should also be time for recreation. His movements should be controlled—he should be under discipline to such an extent that he should have to get permission to do anything out of the routine. Discipline acts as a balance wheel, and as contrasted with complete personal freedom is in the part the secret of the improvement of patients who simply live in institutions.

Correction of Ocular Errors.—The writer can by no means place a high estimate on the value of the correction of muscular and refractive ocular errors in the treatment of epilepsy, and would even express his doubts as to the curative or arresting power of these procedures in any but occasional cases. The ophthalmologist is, however, very important in the treatment of epilepsy; in the first place his expert examination of the retina is of value in determining the presence or absence of tumor; the retinal vessels also indicate something of the state of the cerebral circulation; and determination of the color fields is of value possibly in determining slightly increased intracranial tension and in the diagnosis of hysteria. Even though muscular and refractive errors are not directly causal in most cases, they are one of the variable quantities which added to others make up the etiology of the seizure. It must be our object to remove as many of these causes as possible in the hope of so reducing the value of the left side of the equation to epilepsy that it shall no longer be equal to the right side, *i. e.*, to the seizure. Correction of refraction errors often adds greatly to the comfort of the patient; in other cases it brings improvement of disposition or

diminution of the number of "jerks" as the myoclonic twitchings are commonly called and in an occasional case will stop the seizures. The fitting of glasses and the correction of muscle defects is an aid but by no means a panacea in the epilepsies.

It may be practical to note that the glasses worn by epileptics are unusually exposed to accident, and therefore should have as stout mountings as is consistent. The spectacles with metal rims about the lenses are probably the best; the position of the lenses should also be frequently adjusted.

The ear and nose are also to be examined. The direct relation of nasal conditions to epilepsy has been rarely demonstrated, but, on the other hand, the relation of middle ear and mastoid conditions to a later developing epilepsy is not infrequently proved. It is probably too late, however, in such cases to do more than prevent further damage.

Effect of Treatment.—In estimating the success of any mode of treatment the greatest conservatism is required. A cure cannot with certainty be pronounced until the individual is dead of some other disease and without any recurrence of seizures. "Arrests" may be spoken of, qualifying the report by stating the length of time for which the cessation of seizures has endured. In some cases seizures are comparatively infrequent and intervals of several months not unusual. One must also be cautious about attributing to some one remedial measure all the benefits accruing in a given case. It must be remembered that while a special mode of treatment is being tried, patients are usually under better conditions, and especially under closer observation than ordinarily, so that the hygienic conditions alone tend toward improvement. Careful controls should always be made. Lastly, it must be remembered that almost anything done for an epileptic will bring temporary improvement.

All manner of therapeutic and etiological experimentation in epilepsy has a great underlying defect, that a perfectly homogeneous group of cases is almost unobtainable. Not only are there many individual peculiarities in each case, but we also lack a knowledge of the types of the disease of sufficient accuracy to group them on an etiological basis. As epilepsy is the result of a summation of causes, so must the result of treatment be obtained as the result from a number of simultaneously applied remedies or methods.

Treatment of Emergencies.—Epilepsy is a disease which is characterized by emergencies. The acute manifestations of the disease require special care, and that this must be prompt and energetic, but at the same time skilful, cannot be too strongly emphasized.

The simplest emergency in the disease is the seizure itself. This needs little treatment so far as the individual isolated seizure is concerned. If the patient is observed during the aura he should be placed in a chair or on the ground, etc., so that the risk of a severe fall will be obviated. The clothing about the neck is to be loosened, so that breathing will not be impeded. Such precautions as may be should be taken to keep the epileptic out of the wet and dirt during the attack.

In the clonic stage, some epileptics strike with their hands or feet, or even their heads against the ground, but beyond controlling such movements as promise harm, the convulsion should not be restrained. After the convulsion the clearing out of the throat is facilitated by turning the head on one side or lowering it; this is especially important if vomiting has occurred, in order to prevent aspiration of foreign matter into the lungs. The patient should not be allowed to pass out of observation until he is sleeping quietly or is reasonably himself once more; in these postepileptic states of confusion, the patient is apt to wander away.

Increased frequency of seizures divides itself into two groups—serial seizures and status epilepticus. The former consists of a number of seizures which come at intervals markedly shorter than normal to the individual case, and are accompanied by more or less prostration. What might be a normal frequency for one case might constitute a dangerous series in another case.

Status epilepticus is a far more dangerous condition and is characterized by practically continuous seizures—technically there should be no interval between the individual attacks, but one should merge into the next. Two dangers associate themselves with these conditions, namely, exhaustion and pulmonary edema. Stop the seizures with as little drugging as possible, but stop them. This should constitute our guide in the treatment of these conditions. The condition of the patient when first seen in such a condition is some index as to where to begin our treatment. If exhaustion is setting in it probably will be wise to use a powerful sedative at once, getting the seizures under control in this way, to give time to attack the cause of the condition.

A knowledge of the individual case is of the greatest value and will often save valuable time which might otherwise be lost in trying milder remedies. In cases of mild series or status the condition will often respond to a half-ounce of the following mixture, per rectum:

R—Chloral hydrate	5.0 parts
Potassium bromide	6.6 parts
Aqua	ad. 100.0 parts

This may be repeated in a half-hour if it does not prevent further attacks or they have not been otherwise stopped. A plain enema should always be given to start with and should be repeated at frequent intervals as long as the seizures continue; this cleaning out of the lower bowel is of the highest importance and undoubtedly will of itself stop many cases of seizures. If the patient can swallow it is well to start purgatives by the mouth, so that the clearing out shall be as complete as possible. The hot pack is another simple but usually quite effectual remedy—one cannot ordinarily wait for the water to be heated, but where the materials are promptly available the heat may control the seizures. If enemas and packs and the chloral mixture have failed we must turn to chloroform, giving enough, even up to full anesthesia, to stop the seizures. Cases vary very much in their requirements as

to sedation. With some the first whiffs of chloroform are sufficient to stop the convulsions, but with others only full anesthesia will suffice. Occasionally a case of status will be found in which the seizures continue in spite of full anesthesia. It may be noted that in some cases the locking of the respiratory muscles by the seizure may be so tense that little chloroform can be inhaled; in such instances resort may be had to the unusual method of giving the chloroform by artificial respiration. In extreme cases, where all else in the drug line fails, hyoscine may be tried, giving $\frac{1}{100}$ grain every fifteen minutes up to a maximum of $\frac{1}{25}$ grain. Naturally, this method is only to be used in extreme instances and with due regard to the condition of the patient's heart; hyoscine is a dangerous drug unless used with extreme care.

Just as we have pointed out in connection with the bromides in the routine treatment of the disease, we must also point out that sedation in the emergencies of the disease is a palliative, though also a life-saving procedure. While sedation carries the patient over the first part of the emergency the other methods mentioned above, hot packs and notably clearing out the bowels, are to be used for all they are worth. The hot pack is probably palliative, but the evacuation of the bowels is distinctly curative. In some cases along with the enemas, apomorphine may be given to clear out the stomach. Where there is reason to fear recent overindulgence in food as the cause of the condition this is especially appropriate; care must be taken that aspiration of the vomitus into the lungs is not favored by the position of the head. Venesection is also of value and may be advantageously combined with the administration of saline solution intravenously. Continuous saline administered by Murphy's method per rectum in the intervals between enemas will also be of value. Lumbar puncture is also recommended in obstinate cases of status; frequently considerable clear fluid is obtained.

The exhaustion states after numerous seizures present a serious danger to life and require careful nursing; rest is usually the best remedy. Strychnine may be given to stimulate the heart. Salines either hypodermically or per rectum are again of service.

Pulmonary Edema.—The second complication, pulmonary edema, not only often follows status and series but may also occur after isolated seizures—in fact, the latter is the most fatal form, since a single seizure does not attract the close observation of the patient which is required by serial attacks or status. Acute pulmonary edema is the immediate cause of death in a great many of the "found dead" cases among epileptics; as said before, the patient may drown in his own secretions, while lying quietly on his back. It is difficult to impress on those who have not encountered this condition its true seriousness; writers on the general subject of pulmonary edema mostly fail to mention the epileptic variety, while writers on epilepsy mostly fail to regard it as other than a curiosity. To those who have seen large numbers of cases, pulmonary edema is an extremely serious and fatal condition.

In the majority of cases there is hypersecretion of mucus, as

evidenced by the froth about the mouth; it is questionable whether this is the initial stage of an edema of the lungs, for the edema fluid is not mucoid but is a transudate from the blood. Autopsy in fatal cases often shows congested lungs from which fluid may be expressed as from a sponge; a dilated right heart is a usual accompaniment.

The treatment of pulmonary edema is best prophylactic—stop the attacks as promptly as possible. Atropine has the greatest action of any drug in this condition; it may also be used as a prophylactic, where the onset of the edema is feared or where it is observed in an early stage. After the condition is established, mechanical drainage by lowering the patient's head over the side of the bed, or in extreme cases by lifting him by the heels, will relieve the lungs of much fluid by gravitation. Venesection is also employed and will relieve the congested veins. Counterirritation by dry cupping is also of value. Oxygen should be administered when the embarrassment of respiration is great.

Pneumonias often implant themselves on the favorable soil offered by an edematous lung, and are by far the most serious of the non-epileptic conditions seen in epilepsy and are the chief of the causes of death. The treatment of these pneumonias is not within the scope of this article, and the writer will only point out the seriousness of these conditions and the probability of their occurrence through exposure or through the lowered resistance of the pulmonary tissues.

The *acute mental states* which occur in epilepsy are to be treated in much the same manner as are the convulsive manifestations. In no case should the individual be restrained, either mechanically or chemically, without other treatment. A hot pack or continuous hot bath will probably quiet most cases; in the simple confusions these are not ordinarily necessary as close observation to prevent wandering away will suffice. But in any and all cases eliminative treatment must be promptly instituted and persistently continued, even for two or three days, unless the case clears up. At the risk of being regarded as riding a hobby the writer would reiterate the effectiveness of intestinal evacuation and general eliminative measures in the treatment of epilepsy in all its phases. The whole etiology of epilepsy is by no means contained in the digestive tract, but this region presents a very profitable field for treatment.

Cases presenting definite and prolonged *prodroma* are of importance and are to be here considered. Treatment at the time of threatened attacks can by no means replace a regular daily effort toward the betterment of the case, but at the same time our best efforts fail and a seizure often threatens. Prodroma in the form of motor phenomena—myoclonic movements—or phasic manifestations—irritability, anxiety, and even euphoria, or increasing anesthesia about the upper trunk and sometimes head—all point out to the experienced observer the approach of a seizure. Here, as elsewhere, experience has shown that the thorough clearing out of the alimentary tract, with restricted diet, is better treatment than sedation.

Improvement will follow almost any change in the treatment of the epileptic and, for a time, the new line of therapeutics will seem to be very successful; in the course of a longer or shorter time, however, the patient will be back in his ordinary condition unless the treatment employed has genuinely influenced the disease. Attacks of the infectious diseases seem to exert a sort of favorable influence, and often after them there will be periods of freedom from attacks of varying duration. We do not understand the mechanism of these improvements and cannot use them as therapeutic measures.

Surgical Treatment of the Epilepsies.—Accidents are the cause of the largest part of the minor surgical work among epileptics, and of a good deal of the major surgery also. Except to again urge the continual observation of the epileptic in order that he may not expose himself in dangerous places, and to emphasize the seriousness of the seizure and its attendant unconsciousness as predisposing to serious injury, the treatment of accidents among epileptics needs no special discussion.

FIG. 16



Gross anatomical lesions associated with epilepsy. Tumor of temporo-phenoidal region.

The treatment of the injuries is exactly as in normal people. In only one way is special precaution necessary beyond ordinary work; it must be remembered that the patient may have an attack or become confused or violent, and in this condition tear away dressings and undo the work of the surgeon before the natural union of the parts is sufficiently strong. Both the closing of a wound with stitches and the dressings applied should be strong enough to withstand considerable strain.

Surgery plays a large part in the therapeutics of the disease. Almost every conceivable operation has been recommended and performed on epileptics in order to cure the disease. Unfortunately, in many cases discrimination has not been used in selecting cases, especially when the matter is considered from the viewpoint advocated here, that there are many different epilepsies and that epileptics do not

PLATE XVI

Fig. 1



Gross Anatomical Lesions Associated with Epilepsy: Tumor.

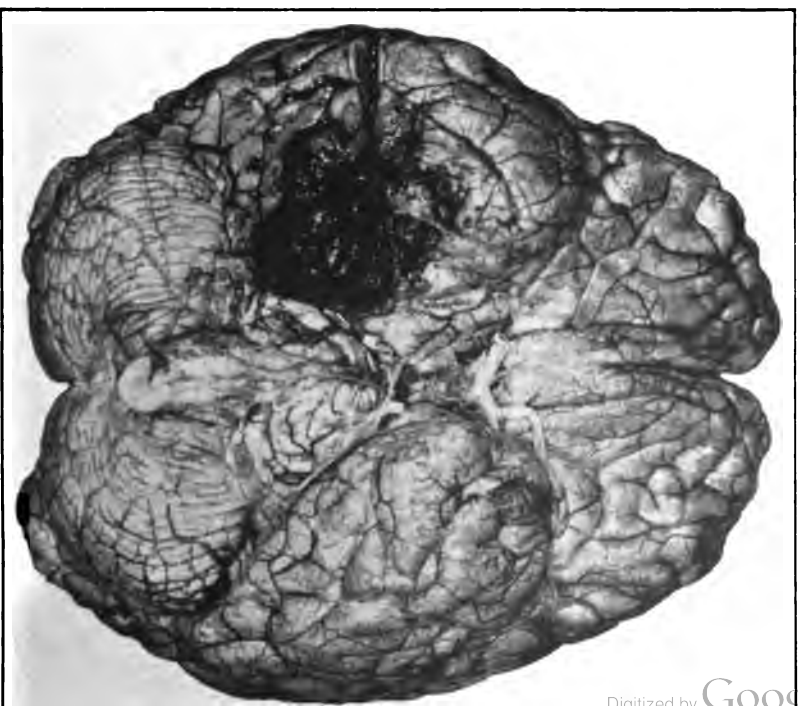
Fig. 2



Gross Anatomical Lesions Associated with Epilepsy: Tumor.

PLATE XVII

Fig. 2



Gross Anatomical Lesions Associated with Epilepsy.

Tumor of temporoparietoparietal lobe, with terminal
hemorrhage.

Fig. 1



Gross Anatomical Lesions Associated with Epilepsy.

Tumor and aneurysm of vertebral artery.

by any means represent a homogeneous material. This fundamental defect is seen everywhere in connection with epilepsy, and can hardly today be remedied as we do not know enough about the kinds of epilepsy to divide them properly.

Surgical intervention in epilepsy is based on one or another of the following conceptions:

1. That seizures which show well-marked focality are due to definite and circumscribed lesions in the cortex, which can be removed with the knife. This, of course, includes cases of epilepsy arising from tumor and from trauma; it also includes a number of cases in which the seizures are limited to a definite muscle group, or else have such a definite point of origin that an assumption of a focus of cortical change is justified.

2. Operations for the relief of intracranial pressure by simple decompression or by fenestrating the dura.

3. Operations on parts of the body remote from the brain, with a view to removing sources of peripheral irritation.

4. Operations designed to make procreation impossible. The discussion of these will be deferred to the section on eugenics.

The object of any operation in epilepsy must be to remove an irritating pathological condition completely, without the formation of scar material which shall continue the irritation.

Operations for tumor we may dismiss with only a word, since the principles of their treatment are so well known. Two points may be emphasized, however, the necessity for early recognition and removal of the tumor and the clear understanding that removal of the tumor may not cure the epilepsy; a tumor causes compression and displacement of brain tissue with resulting disturbances of nutrition; if these have been prolonged, chronic changes may have set in and the removal of the tumor or relief of the pressure cannot be expected to cure the chronic changes. Too positive assurance must not be given a patient as to the result of operation for tumor; the operation should be done if the tumor appears at all operable, but cure is another matter.

Operations in traumatic cases are mostly open to one serious criticism—that if they are needed at all the proper time for their performance is immediately after the injury or at least before the patient has become epileptic. The operation in traumatic cases should be preventative rather than curative.

In deciding the question of operation in alleged traumatic cases it is not alone the focality of the seizures which should be considered, but rather the whole case. In the first place the diagnosis of traumatic epilepsy is often to be seriously questioned or modified; there are cases where the trauma appears to have occurred in the first seizure; hereditary factors are so prominent in others as to make the role of the trauma of importance as an exciting incident rather than as a direct cause; similarly, alcoholism, the effects of infectious disease, etc., when present are to be regarded as modifying the prime importance of a trauma. In many cases the alleged accident is trivial in character

as compared with the supposed effects, and sometimes the epilepsy does not follow until the lapse of an absurdly long time; it must be remembered, though, that the repair of hemorrhages or of encephalitis may be slow and that the resulting repair tissue may be tardy in setting up irritation. Early operation is important.

Leaving out of consideration tumor and traumatic cases the greatest conservatism is to be used in operating with the expectation of finding focal lesions. In a long series of autopsies, about 250 personally performed and about 90 more from the records of the colony—the number of cases in which pathological focal lesions were found was very small, so that operations on these cases with the expectation of removing a focus of pathological change would have been largely disappointing. The excision of a portion of the cortex, corresponding to the muscle group involved sometimes is successful, but must necessarily run the risk of leaving, in an already hypersusceptible region, a scar which may continue that hypersusceptibility. The writer inclines to the belief that the origin of the convulsion in definite muscle groups and sometimes the limitation of the seizure to such a group is rather due to the low threshold for the activity of this particular group of cells—a lowered resistance to noxious stimuli—rather than to actual demonstrable pathological change; he believes, therefore, that operation in such cases, and especially in those cases where grand mal seizures continue or accompany the focal attacks, are not always justified.

The results of a number of trephinations have been shown at autopsy; technically good results and bad had alike died epileptic. In some the amount of adhesion and even cortical atrophy were so great that the operative result must have been far worse than the original state.

Operation for Reduction of Intracranial Pressure.—The operations which aim to reduce the intracranial pressure by raising a bone flap or by also incising the dura and permitting the exit of fluid are better supported by findings at autopsy. Edema of the subarachnoid space is very commonly present. Localized edemas such as are mentioned by Alexander¹ have not been seen either at autopsy or at the operation performed at the colony. The amount of fluid under the dura and arachnoid is sometimes very considerable, but marked signs of increased intracranial tension are not commonly observed in epileptics on a par with those of tumor. Cushing finds that certain changes in the visual fields for colors are present in the slighter degrees of increased intracranial tension, and these may be of valuable diagnostic aid in this condition.

The operation consists of the elevation of a large osteoplastic flap, usually over the motor and adjacent regions, with multiple incisions through the dura and if necessary through the arachnoid, care being taken not to cut bloodvessels in the latter. As much fluid as possible is allowed to drain away, after which the dura is sutured and the flap

¹Lancet, Sept. 30, 1911, p. 932.

returned to place. Some do not return the bony flap, but it seems undesirable to leave exposed so large an area of brain surface, especially in an epileptic who may at any time fall and strike this particular spot.

These operations certainly yield some results. Part of this improvement must be discounted by the fact that "doing something" will bring improvement of itself, irrespective of what is done. There seems, however, to be some benefit conferred by the drainage operations. It would seem, however, that if drainage be the object that some measure should be used to make the drainage continuous; the incisions in the dura soon close and the replaced flap knits into place. The repair tissue may have a higher permeability for the intracranial fluid, but this seems a doubtful reliance. Operations to make a permanent drain have so far been unsuccessful.

It seems an almost unnecessary precaution to point out that the brain is a delicate structure and requires exceedingly delicate manipulation, yet the writer recalls the instance of a man who reported thrusting his finger through the cortex into the lateral ventricle. The brain is not to be abused. It is the object in operating not to injure it but rather to conserve it, and to remove the effects of disease rather than produce them.

Operation for Removal of Peripheral Irritation.—Operations on other parts of the body are frequently performed, with a view to removing a peripheral irritation which is having an unfortunate influence on the case. Naturally it is a good general principle to adhere to that the individual should be placed in the best possible physical condition, and that this should be done by operation if necessary. Peripheral causes undoubtedly play some role in isolated cases of epilepsy. Scars or benign tumors of the skin may be associated with the aura; abdominal or pelvic adhesions often cause great distress and should be corrected. The relation of adhesions to constipation is probably of importance; the presence of Lane's bands and Jackson's veil is often observed in our cases; sagging of the transverse colon and angulation of the sigmoid are also commonly present. Ear, eye, and nose conditions have already been spoken of. Tonsils and "adenoids" often require removal. Castration has been advised in both sexes, but especially in women, on the ground that there was a definite connection between an increased number of seizures and the menstrual periods. Removal of the ovaries has not yielded the results expected, and careful observation does not seem to support the connection between the menstrual period and an increase in the number of seizures. Circumcision in both sexes has been practised, and in females the excision of the clitoris was for a time strenuously advocated. None of these operations are for routine application; the needs of the individual case must always be considered; and while in selected cases the operations may benefit, their indiscriminate application will cause their true value to be overlooked. As far as masturbation is concerned it is a question whether it is not oftener a symptom of a pathological mental state than it is of anatomical conditions requiring intervention.

Surgery alone is not the cure for the epilepsies. It can often relieve one of the quantities which enter the sum of causes which produces the disease, but without attention to the other aspects of the case surgery alone will not cure. Caution must be used lest enthusiasm lead to premature conclusions as to arrest of the disease. From a year to two years should elapse before we begin to talk of an arrest.

Institutional Treatment.—The general requirements of the successful treatment of the epilepsies are, in general, early and careful diagnosis; a mode of life which combines as far as possible the best of hygiene with the best of medical care; discipline, limitation of food, both as to kind and variety, work, exercise, all enter into the treatment.

The writer believes that these objects are almost impossible of attainment in the average home; that while the financial aspect of providing the treatment is not always serious it is the environment of the case which presents the most serious difficulties. Either from ignorance or from mistaken love and sympathy for the patient a strict observance of the physicians' orders is a difficult matter in the average home, especially where this obedience must be extended over a period of months and perhaps a couple of years. Relatives will rarely properly discipline the members of their family; the doctor cannot be on hand at all times to enforce the routine of treatment, and the result is that no matter how efficient the treatment may have been in conception the execution has completely destroyed its efficiency and the patient passes into the group of chronic epileptics.

We have already discussed the results of the disease upon the earning power and general social relations of the patient and have pointed out that these deteriorate in proportion as the disease advances. The mental status of the patient and the results of this deterioration on his personal habits make him undesirable in the home and difficult to endure even there; in addition he is a financial drain, both for his support and for the medicines he consumes. Lastly, the sexual activities of the epileptic at home are uncontrolled.

Based on the social and economical effects of the disease, as well as upon the superior medical benefits accruing, the majority of those interested in this class of dependents are advocating the treatment of epileptics in public institutions built for epileptics only and on the plan of farm-colony hospitals.

History.—So far as the writer can learn the first separate care of epileptics was undertaken at La Force in Dordogne, France, not far from Bordeaux.¹ John Bost, a dissenting Protestant clergyman, founded at this place, through his own personal efforts, six houses built between the years 1848 and 1881; all were for the care of unfortunates, and the third one built was for young girls who were epileptic. While much is owed to John Bost for simply initiating the separate care of epileptics we really owe to him a larger debt for originating the care of these people on farms in the country. Working from

¹ The Epileptic and Crippled—Charity Organization Series, London, 1893.

religious motives he incidentally founded a system which has proved of the utmost value.

From the school for Deacons near Hamburg, Germany, many Home Mission workers have been sent out, and from their labors, among other things, have grown up institutions for the care of epileptics, notably the colony near Bielefeld.

On February 21, 1866, some people in Bielefeld purchased a little farm-house and some land. Delayed by the war it was on July 4, 1867, that the first house-father was installed, and on October 4 of the same year that the first four epileptics were received. General Superintendent Wiessman initiated the work which Pastor von Bodlschwingh took up in 1872 and carried on until his death. The institution at Bielefeld has grown beyond its former sphere of usefulness in caring only for epileptics and now has a wider field of activity. Its greatest work has been the inspiration which it has sent throughout the world, leading to the foundation of many other institutions for epileptics.

The institution at Bielefeld has been the prototype after which all others have been patterned; an extended description of its manifold activities—chief of which is making a place for the epileptic where he can be useful in spite of his disease—would be interesting, but would require too much space. Two cardinal features must be mentioned, the non-institutional atmosphere and the industrious contentment of the inmates. These are ideals to be sought in every similar institution.

Another early institution for epileptics, which made no imprint on the work, was founded at La Treppe. There had been an old custom by which epileptics came to this place each May and September to receive from the Larnage family the free gift of a remedy made from *Galium album*. In 1857 it occurred to the Compté de Larnage, while continuing the free gift of the remedy, to found an institution for the maintenance of epileptics, which was continued under the charge of a Catholic sisterhood.

In Germany many colonies and divisions in other institutions grew up for the special care of epileptics. In England the Maghull Home (1889) and the Meath Home (1893) were the first; there are now several others, notably the colony at Chalfont St. Peter, which is maintained by the National Association for the Employment of Epileptics; the colony of the London County Council, near Epsom; at Langho, Lancashire; the David Lewis Home near Alderley Edge, Cheshire, and others.

The Ohio State Board of Charities has the proud distinction of having been the first in America to recognize the need of special provision for the epileptic, having made such recommendation as early as 1868. Its efforts bore fruit in time, and on August 7, 1893, the institution at Gallipolis was opened, the first in America. The New York State Commissioner in Lunacy advocated the matter in 1874 and in 1878 the State Board of Charities added its approval. The immediate efforts of two men, Dr. Frederick Peterson and the late Hon. William Pryor Letohworth, had most to do with the crystallization of sentiment

which resulted in the founding of the Craig Colony for epileptics; the State Charities' Aid Association also lent valuable aid. The first patient was admitted January 27, 1895. These institutions in Ohio and in New York were pioneers in the work in this country; other States have followed, and now New York has a second institution and Ohio is considering of another, and the following States have also founded colonies: Massachusetts, New Jersey, Texas, Kansas, Indiana, Virginia, Connecticut. In Pennsylvania there are two semi-private charities for the epileptic, and in Missouri there is also one.¹

Institutional Standards.—The special institution for the epileptic has to meet two general demands: (1) It must supply the expert treatment and the proper environment to make that treatment effective—it must be a hospital; (2) it must receive and care for those chronic epileptics who can no longer support themselves or who can no longer be kept at home. There are thus to be two distinct divisions in an ideal institution for all classes of epileptics—the hospital and the custodial.

The institution should be reserved strictly for the epileptic. Experience shows that it is not practicable or desirable to treat the epileptic along with other public charges, as they tend to clash and to irritate one another. The seizures of the epileptic are very disturbing to the non-epileptic while practically unnoticed by the other epileptics. Institution men are a unit as to the advantage of removing the epileptic from contact with other State charges, and report that where this has been done that great benefit has resulted to both classes. The epileptic is a different problem in many ways from the feeble-minded or the insane, though he often also belongs in these groups—a special institution best subserves the interests of all.

Eugenic Factors.—The proposition of caring for all epileptics is one which appeals strongly from the standpoint of eugenics, but which from the practical standpoint can probably never be realized. Segregation would of course limit the ability of these people to have children. From a much narrower point of view—that of what is practicable—we must probably limit ourselves to caring for the chronics who can no longer “float” in outside life and to treating recent cases. It is the financial side of the question which makes it necessary to limit the care of epileptics, though as someone has remarked it is in the end more expensive not to segregate them than it is to maintain them for life. The undertaking would be very great, especially when we add to them the number of the feeble-minded and psychotic; the criminal, the drunkard, and the tramp will also have to be considered. The lowest estimates assume one epileptic in each thousand of population, and of these at least a quarter can no longer “float” and demand institutional care, and another quarter (recent cases) should be receiving

¹ For a mass of valuable data as to the care of epileptics in public and private institutions throughout the world, consult Letchworth: *The Care and Treatment of Epileptics*, Putnam, 1899; *Transactions National Association for the Study of Epilepsy*, vol. i, and following volumes.

hospital care. The writer is an advocate of public care for every epileptic for whom provision can be made, especially in view of the segregation of the sexes obtained.

Objections.—There are certain objections to such a course, some of which will be briefly stated:

1. The stigma of having been colonized or having a relative at a colony is referred to. The real stigma is not that an individual is an epileptic or that he is receiving treatment, but lies rather in the fact that the family stock or the patient's life is such that epilepsy has occurred. Of course, this would not apply in genuinely traumatic cases.

2. That State care is undesirable paternalism, relieving parents of a responsibility which they should not be allowed to escape easily.

3. The effect of seeing many others who are suffering from the same disease and the possibility of mimicry. Mimicry is very rare and the epileptic is usually sympathetic, with other epileptics, and is not unpleasantly influenced by their seizures; the only exception might be patients of high mental grade who perceive the downward drift of other epileptics and fear the same condition. Such cases are rather exceptional. The mental dulling which accompanies the disease usually prevents such comparisons.

4. It is urged that mild cases can be and are successfully treated at home; that colonization or hospitalization will make the general practitioner and family physician anxious to shift his responsibility, and will lead him to be careless of his efforts for the epileptic at home. Institutional cases and the conversation of physicians seems to controvert this claim. The patient is not commonly successfully treated at home, at least from a curative standpoint; many are kept along on bromides in a fairly comfortable condition, but these soon pass into a chronic state. The difficulties of carrying out any adequate treatment at home make it almost impossible that the physician do the work no matter how well he is instructed.

Character of Institution.—A few points in institution design may be mentioned. Stiffness and uniformity of design are to be avoided in colony construction, as these qualities make for the institutional atmosphere. In the ideal the buildings are diverse yet harmonious and scattered about in such a way as to reduce to the minimum the prison-like regularity seen in some institutions. The writer will not soon forget his first view of Bielefeld—it was the impression of an ordinary village rather than that of an institution. Of course, there are always parts of an institution which are hard to disguise, yet by utilizing the natural physical characters of the site and a suitable diversity of design much can be done in this direction. The effort should be made to create the impression of a rambling country village in which a modern hospital, shops, etc., have been built. A non-institutional atmosphere is of great service in creating the home-like surroundings which are necessary to make the colony successful; the patients must be contented, and they will be more so in a home-like atmosphere.

The colony should be divided into two main divisions: the hospital division and the "homes"—the latter term being preferred to "custodial." In the former, cases of recent onset should be received as soon as possible after their disease begins, whether infants or adults; they should remain for a shorter or longer time according to the case—probably a year will be the average, so that the capacity of this division cannot be small. It should be equipped in every way as a hospital, mostly for medical cases, but with ample provision for surgical work; the medical and nursing force should be numerous, for individualization of the treatment requires a plentiful corps of workers. The patients in this division should work; they are not to be regarded as invalids, but work is to be assigned as part of the treatment. If after thorough trial it does not seem possible to permanently benefit or cure the patient he should leave this division and preferably enter the "homes;" many will probably go to their own homes, on account of lack of room to care for them. In these "homes" an attempt is to be made to make a little social system suited to the needs of the epileptic: comfort, work, regularity of life, amusement and religious facilities must all be provided. The village must be complete. Here the epileptic should live, working his maximum, his disease curbed by observation and treatment, ending their days in the "homes." Unless the attacks are actually arrested for a long period the writer does not advocate the return of the epileptic to the outer world. Such return often results in a relapse, and there is, too, the serious question whether even the cured epileptic cannot still transmit to progeny the hereditary characters which make defectives. Rather should he remain to fill a useful place in the village.

A third division should be provided for the cases of such low mental epileptic grade that they are absolutely hopeless. Possibly another building or division might be used, namely, a division for the psychotics.

Provision must also be made for the tuberculous epileptic. It appears that the epileptic is rather sensitive to pulmonary infection, and buildings for the isolation and treatment of these cases should be erected. It is probable that the frequent slight congestions and edemas following seizures create a soil favorable to the disease.

Cost of Institutions.—While institutions for epileptics are to be considered in the main as either medical or as custodial, it must not be forgotten that they are also to be labor colonies. The cost of maintenance in an institution is approximately the same for the epileptic and for the psychotic, the exact figure depending on the system of charges and deductions used in the book-keeping of the institution; but the cost will be between \$175 and \$200 per year per capita under favorable circumstances. The epileptic cannot be made self-supporting or at least the average chronic case cannot; it is because of this that he seeks care. He can rarely do a normal day's work, both on account of his seizures and because of the mental deterioration. Nevertheless, almost every epileptic may contribute more or less labor, the value of which will aid in reducing the gross cost of his maintenance. Many can do

no more than house or kitchen work; some can do no more than, for example, push a floor polisher; a few do excellent work at trades. Dr. Spratling, who always took an optimistic view of epileptic labor, placed the average yearly value in labor of institutional epileptics at \$30.¹

In connection with work for the epileptic we meet again the variability of mood already mentioned and the necessity for the exercise of tactful and comprehending management. It seems to be difficult for many patients to keep steadily at work. This is due partly to their disease, but in large part it is due to the attitude in which they have been encouraged before coming to the colony. They come to look on themselves as unfortunates, so much to be pitied that society owes them maintenance. This is a wrong attitude. Sympathy is no reason why, because an individual is epileptic or defective, he should be supported in comfortable idleness as a guest of the State. While the epileptic is to some extent an invalid he is usually fairly robust physically and capable of some work. The charity of our institutions is abused because public sentiment regards the epileptic as an invalid and unfit to work; the institutions can neither require work nor successfully dispose of what product they have on account of legal restrictions.

A few words may be added as to some of the physical characters of the buildings to be erected. The site of the colony should be sufficiently isolated so that the patients will not have easy access to a town; but, on the other hand, there must be transportation by which the employees may get away without too great difficulty. The buildings must be built with the seizures of the disease in view; corners should be rounded, stairs moderate in slope and with high rails, and no long flights. All hot pipes and fires should be adequately protected, and the screening must be far enough away from the piping so that the fingers cannot be thrust through to the hot surface. The setting of the kitchen stove in a recess which just fits it greatly reduces the possibilities of injury. The higher the mental grade of the patients the smaller the building in which they should live—20 high-grade and 50 low-grade cases represent the extremes. In the buildings for the "hospital" cases there must be provision made for treatment rooms or for easy access to such rooms; it is not perhaps necessary that the "hospital" division be constructed after ordinary general hospital plans, but rather the convenience of the general hospital arrangements should be combined with the comforts of small cottages. A hospital building for acute and for surgical cases is, of course, necessary, and similarly there should also be a building for the mentally disturbed.

Scope of Institutions.—There is some difference of opinion as to the scope which an institution for epileptics should have. Personally,

¹ Based on the average daily population for the past fiscal year the home product of the Craig Colony was worth between \$47 and \$48 per capita. This amount is gross and will be reduced by the charges for supervision, etc.; but it also represents the average on *all* the patients, a considerable proportion of whom are of such mental grade as to be non-productive.

the writer believes that by utilizing advantageously the conformation of the site, divisions for the curable, for the custodial (homes and custodial proper), and for the psychoses can be profitable included in one institution. The objections to such a combined institution which are raised are largely inoperative, and the transition of our cases from normal to psychotic or demented makes the composite institution very desirable.

To sum up: the difficulties of home treatment, the handicap which the patient has to meet in self-support, and the advantages which he can receive in an institution make institutional treatment of the disease most desirable.

Legal Status.—The epilepsies are important medico-legal conditions, as the mental states and mental deficiencies which accompany them sometimes bring the epileptic into the hands of the law.

Criminal acts are not uncommonly committed by epileptics, and these acts may be classed in two groups:

1. Acts done within the interparoxysmal period. In these cases, there is no question of a dimmed mental state or of automatism; the whole question legally, in New York State at least, is whether the individual is mentally capable of understanding the nature and quality of his act. From this standpoint the determination of the amount of mental defect is of great importance. The methods have been hinted at earlier in this article; it is a difficult matter to make a jury accept the estimate placed on an accused by means of scientific testing, especially if the accused has been able partly to support himself. The work of the juvenile courts shows that the juvenile delinquent is commonly a defective; that these children lack the mental capacity to know that a given act is right or wrong. The same is true of a great number of epileptics and the offences they commit in the interparoxysmal period; their minds are so enfeebled that there is nothing to properly curb them in carrying out their desires or the suggestions of others. For acts of this sort one does not think of punishment but rather of training in right and wrong, together with a restraint which will make their uncontrolled actions less dangerous.

2. Numerous states of altered consciousness occur in epileptics, in which they are more or less completely unconscious of what they are doing; acts done in these states of unconsciousness or automatism are usually characterized by violence. The existence of such states is not infrequently raised as a defence in cases of crime; the epileptic alleges that the act of which he is accused—and in these cases the question of the truth of the charge is not usually so much in dispute as is the responsibility of the accused—was committed while he was in a state of epileptic automatism, furor, somnambulism, etc., that he was acting, involuntarily, unreasoningly, unconsciously, and could therefore have no knowledge of the nature and quality of his act; his normal conscious personality was completely in abeyance, and no recollection of the occurrence has persisted.

In an instance which fell under the observation of the writer the

patient D. had had a mild seizure a few minutes before he was noticed by an employee K. standing at the latter's elbow in a threatening manner. K. recognized the situation and walked away in order to avoid trouble, but the patient followed him in absolute silence and his threatening manner suggested cold, set purpose; the most noticeable features were the staring eyes and set expression. Had this patient had a weapon, or had the employee offered any resistance, a tragedy would have been likely. In the retreat of the employee he crossed a railroad track, and the patient in following tripped and fell and afterward stopped the pursuit. A few minutes later he denied all recollection of the incident and was very sorry that it had happened. To the casual observer the patient was simply a man in an uncontrollable rage, one of the terrible cold passions which affect some people. He acted in every way in a normal and coördinated manner, and only the set, staring of the eyes were noticeable to the close observer.

These actions performed in the automatic state or state of epileptic unconsciousness are marked by some variation from the actions of a conscious individual in the same position. They are usually unreasonable, either through the inappropriateness of the action at the time and place at which it is done or through the quality of sudden and excessive violence. The absence of the normal directing and inhibiting powers are seen in all these conditions—the individual works as a machine would work which had been wound up and set going. In some of these conditions the sexual element is uppermost, as in the case of exhibitionism. The occurrence of the automatic state cannot be predicted; some epileptics always have them after seizures, while in others they are rare or may never have been observed until the incident in question calls the matter to notice. They constitute a very important argument for the close observation of epileptics, and are one of the ways in which the epileptic is dangerous.

Amnesia generally follows these states, and by some the completeness of the loss of memory for the automatic period is a proof of the epileptic character of the condition; these observers believe that the memories of the automatic period cannot be recovered by any means now at our command—in fact, they mostly assume that they are totally lost. This is used as a distinction between epileptic and alcoholic amnesias, memories in the latter being recoverable. It seems very probable that it is extremely difficult and probably impossible to reconstruct the memories of the amnesic period.

These amnesic periods are frequently the defence offered to avoid responsibility for a criminal act. It is difficult to prove or to disprove such a contention, since it is very rarely that the alleged mental state of the accused comes directly under observation, and we rather have to consider indirect evidence of the probability of an automatic state having existed. The accused must be shown to be an epileptic, and his seizures should be observed if possible by experts; where the history of seizures is not clearly obtainable, evidence of nocturnal attacks or "spells" of any sort should be sought for, but not by leading questions.

Evidence of a neuropathic heredity is of some value, but is not of the importance usually given it. Scars and recent wounds which cannot be accounted for as being the results of ordinary accidents, etc., support the idea of a seizure, and if the accused is seen soon after the automatic state he should also be examined for the petechial hemorrhages which sometimes are found about the neck and shoulders and conjunctivæ of patients after seizure; for signs of pulmonary edema; and the clothes should be examined to ascertain if he has wet or soiled himself. If sleeping at the beginning of the automatic state the bed should be examined for any signs of seizure—urination, defecation, flow of mucus or blood-stained fluid from the mouth. A seizure need not be established as preceding the automatic state; it seems probable that such states can occur without any seizure or may be preceded by a seizure so slight that it is not noticed.

The motiveless nature of the act and the excessive violence which is done are additional stigmas of the epileptic state.

It is to be recalled that along with epilepsy there also occur psychoses, and that under the influence of these conditions criminal acts may be done. Similarly, feeble-mindedness is sometimes associated with fits of violent rage; but of these periods there is usually recollection.

The relation of epilepsy to the ability to execute a valid will depends on the status of the patient at the time of the execution of the document. It would have to be shown that the individual was in his ordinary mental state, and that he was neither sufficiently dulled by dementia nor primary feeble-mindedness nor distorted by a psychosis to render him incapable of disposing of his property. As has been said, practically all epileptics show some mental defect, but the degree varies greatly in different cases. The fact of epilepsy should not directly influence testamentary power unless the epileptic were at the time confused or automatic, mentally deficient or in a psychosis.

The status of the epileptic in our institutions is not sufficiently well established. The ordinary epileptic, while at times subject to mental disturbances which make him, from a practical standpoint, at those times insane, is not insane in the ordinary sense, and the normal periods are so long in comparison to the abnormal that it is an injustice to him to commit him to a State hospital. In fact, many State hospitals will not accept such cases. Similarly, the epileptic who is markedly feeble-minded will not be accepted by many of the homes for the feeble-minded, so that he occupies a position which is ill-defined unless there is a special institution open to him. In some institutions for epileptics the residence of the patient is more or less voluntary—in some States he is committed—but there is usually far less control of him than there is of the insane. The writer adheres very strongly to the belief that it is best for the patient and for the public that he be withdrawn from the world at large and be committed to a colony, at least until he is cured. At present, patients grow homesick and persuade their relatives to withdraw them, often before institutional life and treatment have had a sufficient trial.

It should be made legally possible to commit an epileptic to a colony under such provisions as will insure his remaining there long enough to profit by the care given him; in the case of the insane, the feeble-minded, and those showing undue sexual tendencies the confinement should be for life. The latter is especially desirable in the case of women of such low mentality as to make them defenceless and immoral.

The Symptomatic Epilepsies.—In the preceding pages we have discussed methods of treatment applicable to all kinds of epilepsy, methods which are suitable on account of the common characteristics of the syndrome in all cases of whatever kind. These measures are practically the only ones available in the group of cases in which we cannot recognize some obvious cause; it must not be forgotten that we have assumed that all epilepsies are symptomatic, but that in the unclassified or idiopathic group we are unable to name the main causative agent.

Even in the symptomatic epilepsies the condition is probably the result of a summation of etiological agencies—that the obvious one is only the most important of several. Naturally it would seem that such cases should be much more hopeful, since we have something tangible to work on. However, most of the things which cause epilepsy also cause chronic changes in the central nervous system, and it is perhaps an open question whether it is the action of the agent directly on the cells or the result of these chronic changes which causes the epilepsy. The great field for endeavor in the epilepsies, as in all other diseases today, lies in prophylaxis; and the group of the symptomatic epilepsies offers the best field for this sort of effort.

Too much is not to be expected from late treatment, nor can the treatment be confined to any one line even in these symptomatic cases; we have to reach not only the obvious but also the adjuvant causes which lead to the seizures. The later treatment is begun, and the narrower it is the less is the hope for ultimate cure or improvement.

Syphilitic Epilepsies.—Syphilitic epilepsies are perhaps not so common as is sometimes supposed. The lesions seem to be varied: gumma of the brain; vascular weakening and rupture, giving postapoplectic cases; endarteritis, with degeneration of the central nerve cells; and lastly, the action of the poisons of the disease directly on the nerve cells. The desirability of early diagnosis and treatment of syphilis as a prophylactic measure need not be mentioned; fortunately, the Wassermann reaction places in our hands an accurate diagnostic method which permits an early diagnosis. Of the treatment of the syphilis nothing need be said here, except that it should be prompt and energetic and continued.

In salvarsan, with its prompt action, we may have an ideal weapon for combating syphilitic conditions of recent onset. Ehrlich has given as a contraindication chronic nervous disease, but in a few cases of epilepsy treated with it at the colony no harm has apparently been done, and one case was slightly benefited. All were old cases and rather unfavorable subjects for experiment. The warning against the use

of salvarsan in cases having renal conditions may have special application in epilepsy. Chronic changes seem to be quite common in the kidney of epileptics; in general these do not produce symptoms, but in about 20 per cent. of all cases there are found casts and albumin in the urine passed shortly after a seizure. The urine should therefore be especially carefully examined in epileptic patients before giving salvarsan. (See Chapter VII.)

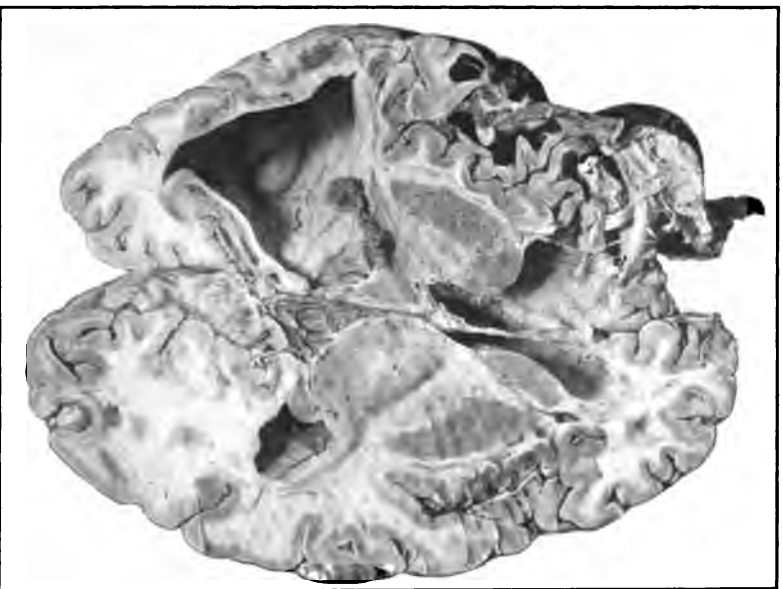
Traumatic Epilepsies.—In no condition do the truths expressed in the equation to epilepsy show more completely than in the traumatic epilepsies, or at least in the group of cases which are so classed. A purely traumatic case is a rarity—almost always there will be found an admixture of heredity, alcohol, sexual excesses, or the effects of some infectious disease. The necessity of a critical diagnosis has already been pointed out in connection with the surgical treatment of the disease. The importance of early operation may be again emphasized—that in every case where there are signs of fracture or hemorrhage, of pressure or of irritation, prompt surgical intervention is indicated. This constitutes a prophylactic measure against epilepsy, and should be carried out rather than to wait until the epilepsy is established and possibly irreparable damage done to the brain. On the other hand, after the disease is established, conservatism in advising operation is desirable. Simply because a patient or his friends say that he sometime fell and hit his head preceding the onset of the disease does not make the condition necessarily a traumatic one; there must be corroborative evidence, and the other factors present must not be of such magnitude as to warrant the idea that the trauma was perhaps only a “last straw.” In these late cases the possibility of improvement by operation must be balanced against the possibility of finding extensive chronic changes, and of the formation of postoperative scars and adhesions which will undo the work of the operation.

Alcohol and Epilepsy.—Alcohol seems to have in many cases an almost specific action on the nervous system of some people, manifesting itself as convulsive activity. In this class of cases no seizure occurs if the individual remains an abstainer, but so soon as he indulges a seizure is the prompt reaction. For these cases, institutional life is of the greatest value because of the associated discipline; these people lack the will power to prevent indulgence when living outside, but the discipline of the institution steadies them sufficiently to keep them straight. In these cases alcohol is either directly or indirectly a specific poison.

Another group of patients are chronic alcoholics. They have been drinking for a longer or shorter time and are thoroughly accustomed to the alcohol. Their epilepsy usually develops late in life (at least middle age) and is not always accompanied by the cardio-renal-vascular lesions which might be expected. It is probable that we have to deal with a chronic alcoholic poisoning of the nerve cells, which has produced the epilepsy. For these conditions there is not much hope of cure unless the condition is very markedly the result of other factors added

PLATE XVIII

Fig. 1



Gross Anatomical Lesions Associated with Epilepsy.
The result of a bullet wound received some thirty
years before death.

Fig. 2



Gross Anatomical Lesions Associated with Epilepsy.
The results of a birth trauma.

to the alcohol. Abstinence from all alcoholic drinks and the general hygienic treatment which we have described is about all that can be done for these cases.

Birth Injuries and Epilepsy.—There are a number of epilepsies which are the late symptoms of damage done by birth injuries, infantile cerebral palsies, encephalitis, or meningitis, and which are generally accompanied by signs of the destruction of some portion of the cerebral cortex, as indicated by a hemiplegia, etc. Birth injuries are probably to some extent preventable, the remedy consisting in the better teaching of obstetrics, so that better diagnosis and treatment of the abnormalities of labor may reduce the number of injuries. Cushing, of Baltimore, has advised operation in such cases soon after birth and reported a mortality of 50 per cent. The present trend of opinion seems somewhat against such intervention on account of the fact that the injury is generally not merely one large clot, but in addition other smaller foci are present which cannot possibly be reached by the knife. On the other hand to contemplate the fate of the weaklings which survive without operation—paralytics, imbeciles, and idiots—makes it seem desirable to take the radical course of trying to do something for most of the birth injuries, especially when one-sided and somewhat localized.

Infectious Diseases and Epilepsy.—The onset of the disease is frequently attributed to an infectious disease, which, while perhaps not directly known to have been accompanied by a meningitis or an encephalitis, nevertheless was really thus accompanied. The results of the repair of these inflammatory conditions gradually become centres of irritation from which the stimulus to convulsion extends. The prognosis in these cases is poor for cure—the damage is done—but general treatment should bring improvement. In addition to inflammatory processes, hemorrhages, both due to violence—as from coughing in pertussis—or from injury of the vascular endothelium by the poisons of the disease, must be mentioned.

In institutional experience, *lead* seems to be a rare etiological factor or at least one which is not often recognized. Where epilepsy follows lead poisoning—probably as the result of an encephalitic process—we can only attempt to eliminate the lead and to keep the individual in hygienic surroundings.

Cardiac Disease and Epilepsy.—The cardiorenal vascular complexes seem to have numerous representatives among the cases with onset in later life. *Arteriosclerosis* is not uncommonly the apparent cause of epileptic attacks, occasionally through the irritation of hemorrhages, but more often, as judged by autopsy findings, from starvation of the nerve cells. When the arteriosclerosis is accompanied by high blood pressures, usually through the addition of a renal condition to the complex, we have a great deal which can be done for the patient. Rest and reduced nutrition, salt starvation, iodides; a little nitrite if the blood pressure is very high; hydrotherapy; but quiet and rest are what we have mainly to rely on.

There was a time when cardiac epilepsies were considered a definite etiological group, but at present this is not the case unless one considers the seizures of Adams-Stokes disease to be epileptic in character. These are due to cardiac asystole and consequent cerebral anemia, and it is probable that those cases of epilepsy in which complete cessation of the pulse has been observed preceding seizure belong to the Stokes-Adams syndrome.

Prophylaxis.—It will be seen that in many of our cases from the very nature of the processes involved, we are hopeless of cure from the very beginning. In others, where there may be hope, we are commonly handicapped by having to deal with conditions which have been established for a considerable time. Occasionally cure, and in most cases improvement, both medically speaking and as regards the manner of life of the individual, sums up what we can do for the epileptic. No matter what the future may show as to the etiology or mechanism of epilepsy it is not probable that we shall be able to restore what the confirmed epileptic has lost or to repair the damage which in some cases has caused the epilepsy. Two things we can do, however: (1) Give early and efficient treatment to the already epileptic, and (2) prevent epilepsy and epileptics.

Prevention is the keynote of modern medicine. It has long been sounded in the infectious diseases, and it has begun to be heard of in regard to the cardio-renal-vascular conditions, in psychiatry, in feeble-mindedness. It is time that a persistent effort is made to study the prevention of epilepsy.

Heredity.—Eugenics presents itself to us first of all in considering the question of prevention on account of the strongly rooted belief that epilepsy is hereditary. We must not, however, limit ourselves to producing a race of normal heredity; it must also be kept in this condition, so that in addition to our seeking to insure the birth of children of normal heredity we have also to consider the removal of all the noxious influences which may enter into human environment. The prophylaxis of epilepsy and eugenics is not identical, but the former includes the latter.

Naturally, as the neuropathic heredity of our epileptic patients presents itself in a numerically striking manner, we seek in eugenics an important agency for its restriction. When we investigate the foundation for this belief in the hereditary character of epilepsy we find that the various authors who have presented data vary very much in the conditions included and in the percentages reported; if we consider what manner of material has been used as the basis of these reports we shall feel less confidence than ever in their value. The data has been mostly drawn from the records of institutional cases, and until recently the defects and general worthlessness of the heredity as recorded in such histories were generally unappreciated. The spaces left in the blank forms commonly used for admission histories are pitifully small and few; the questions they ask represent only a part of the knowledge we should have. Generally filled out by some one

member of a family the facts given are those known to that person. It is well known that most people do not know much of their ascendants in the direct line, and it is equally important for heredity studies to ascertain the manner in which the collateral branches have progressed. In these histories we also meet with family pride—either conscious or unconscious, which tempts our informants to put the most favorable light on incidents or individuals; for example, moderate degrees of feeble-mindedness are not often recorded. A fair-minded survey of the ordinary hospital family history will show that we do not know very much of the family; any attempt to extend this information by correspondence is so laborous that it rarely yields much except in occasional cases. Hence, much of the material as to heredity now on record can only be construed as pointing out the way for further investigation with improved methods.

In other words, while most of us are morally certain of the action of heredity in some manner in producing epileptics we have not the scientific evidence to warrant our formulating so important a conclusion. We *seem* to have the evidence, but, as pointed out, on close investigation it is relatively worthless for accurate and scientific work. From the data at hand it is only safe to postulate that neuropathic ancestry and certain intoxications (*e. g.*, alcohol) in the preceding generation make the individual more liable to epilepsy and to other neuropathic conditions. We do not know why epilepsy appears rather than insanity, etc., in a given case.

A scientific approach to the problem has been made through the efforts of a few men, notably Davenport at the Experiment Station at Cold Spring Harbor, and Goddard and Johnson at the Training School at Vineland, N. J. Through the development of a method they are attempting to bring the data of human heredity within the limits of scientific precision. The method consists in the *personal* investigation of the members of a family and of their collaterals by a trained "field-worker;" this investigator, usually a woman of tact and good education, starts from the heredity data, which the ordinary hospital admission paper gives, and personally sees as many of the family as possible and learns from them all she can about the family and its members, especially in regard to mental status and as to neuropathic conditions; nervous disease, criminalistic traits, feeble-mindedness, insanity, alcohol, etc., must all be sought for. Not only is the family interviewed, but the doctors and ministers who know the family are in turn visited in order to get all the data possible. The results of such an investigation are surprising: (1) the number of individuals who are included is astonishingly large as compared with those included in the ordinary hospital report; (2) the large number of individuals who show some trait which is or may be of importance in neuropathic heredity. Family prejudices are eliminated, an impartial estimate obtained by an observer accustomed to the work, so that the completeness and accuracy of the work present a wonderful improvement over old methods.

Such work is as yet in its infancy; one fact of importance has been elicited, namely, that the mating of feeble-minded parents always produces feeble-minded children. Beyond the fact that epilepsy tends to occur in families in which there is a neuropathic taint, it cannot be said that the investigations have shown any great light on the heredity of epilepsy, unless to reveal an amount of neuropathic heredity greater than heretofore supposed.

The ultimate object of heredity studies is to formulate the laws according to which the taint is transmitted and transmuted, showing itself sometimes in one guise and sometimes as another. The Mendelian law is a type of what is sought. Work must continue along this line until we know these laws; then and only then will we be in position to demand radical measures for limiting the propagation of the unfit. The question of the feeble-minded may be considered as determined—they should under no circumstances mate, and, as many of our epileptics are feeble-minded, the question is settled for many of our cases by a line of collateral research. The epileptic who presents no primary or associated feeble-mindedness (as contrasted with dementia) presents a different problem. The obvious facts lead one to a belief in the efficacy of heredity to produce epilepsy, but when we consider the disease as the sum of the results of several agencies, it seems probable that it is not epilepsy which is inherited, but rather some fundamental liability of the nervous system, which in one set of circumstances yields epilepsy, while in another individual with different environment gives another.

The writer, therefore, ventures to express the doubt whether we shall ever find a law for more than the transmission of this taint; again, since we contend that epilepsy is not a unit condition, but rather a syndrome having varying etiology, it cannot be expected that its heredity will be uniform; the students of heredity in epilepsy will have to meet the same problem which all investigators of the disease must encounter, namely, the non-homogeneous character of the case material studied. It is the neuropathic taint and not epilepsy which will be the important hereditary quantity.

This conservative attitude is adopted not only through belief in its truth, but also because the writer believes that in the long run more will be gained in the cause of eugenics by waiting and educating the laity to the needs of the situation than by forcing upon them laws for which there is no sentiment except among the instructed. We must first have scientific evidence regarding the transmission of these things—evidence which will bear close scrutiny—upon which to base a campaign of education; after this we can enact and enforce laws.

Our standards of humanity do not permit us to leave the processes of the survival of the fittest free to act, nor can we actively aid in the elimination of the unfit. On the contrary, the entire trend of our civilization has been toward better care of the defective and thus the prolongation of his life. With our present standards no other course is open to us; we may speak of euthanasia for these cases as a desirable

solution of the problem, but no one will pass beyond an academic discussion of such a procedure. Instead, we must limit ourselves to the restriction of the propagation of defectives through the enforcement of measures which will make procreation impossible by individuals who are unfit. At present three methods appear available for the solution of this problem of sex isolation among the unfit:

Segregation.—In segregation we have a method which is efficient just in proportion to its completeness. The almost insurmountable difficulty of this method lies in the great numbers who must be segregated; if we consider the number of individuals who come within the groups to be segregated—the epileptic, the insane, the feeble-minded, the criminal, the drunkard, the tramp—some idea of the magnitude of the problem can be obtained, and from a practical standpoint it would appear improbable that any adequate provision could be obtained to meet the needs of such segregation.

Limitation of Marriage.—In the legal limitation of marriage, on the basis of mental and physical fitness, an attempt is made to solve the problem in a different way. It is not, however, sufficiently far-reaching, as many of the most dangerous matings (from the eugenics standpoint) are consummated outside the bonds of wedlock. A few States have enacted such laws, but they are at present more or less inoperative, on account of the lack of a sentiment in their favor.

Sterilization.—Lastly, operative measures to make procreation impossible are advocated by some. Castration, which was formerly advocated for this purpose, has fallen into disuse, on account of the radical changes which the absence of the internal secretions of the testes and ovary may bring about. A simpler operation is used, especially in male subjects, namely, the ligation of the ducts through which the sex cells pass. In the male the operation is very simple: a small incision is made over the spermatic cord on either side and the vas deferens exposed and cut between ligatures; a piece may be removed if desired. In the female the Fallopian tubes may be cut between ligatures in a similar manner. There seems some possibility that the operation in the female may not be certainly effectual through reopening of the passage; as the peritoneum is entered, whether the incision is through the abdomen or through the vault of the vagina, it is perhaps as well to make results certain by removing the ovaries. These operative procedures must be authorized by law, and are usually well provided with mechanism to safeguard the patient from too radical intervention. The decision to operate (castration, vasectomy, etc.) is usually vested in a commission, and sometimes the courts are given direct review of its decisions.

None of these solutions are satisfactory—the problem is too large to be arbitrarily dealt with; and what is equally important, there is no public sentiment to demand intervention. Nor will the segregation or asexualization of *existing* cases of transmissible defect stop the production of defectives. There are influences constantly at work in our civilization tending to pull down the normal to the level of defectives,

and as long as such influences act there will be new defectives, even though the old cases never produce progeny.

Prominent among these deleterious influences attacking the race are alcohol and the venereal diseases. Alcohol and syphilis are probably the source of the greatest portion of all neuropathic taint, which as passed on from generation to generation is added to and magnified. Until we gain control of these agencies our efforts will be entirely on a par with those of the man who bails a leaky boat without stopping the leak.

Education.—The writer believes that a campaign of education based on scientific facts concerning the transmission of hereditary neuropathic characters, and the production of such neuropathic taints in the previously normal, will do more than laws can ever do to prevent the propagation of the feeble-minded. Once the people understand that the psychotic, the epileptic, the feeble-minded, the criminal, the alcoholic, the syphilitic, etc., infect their young with the taint of degeneracy, just so soon will there be a voluntary limitation of the matings among thinking people between those who are tainted. With the frankly defective we can easily deal; for this we require laws of one sort or another—segregation, operation, what you will, so long as they are thoroughly carried out. It is among the border-line conditions that the greatest difficulty will be experienced, and a sentiment which will make people hesitant of taking any chances for the future generation will do more than a thousand laws.

We advocate, then, for the solution of this problem:

1. The scientific study of human heredity.
2. An extensive and sustained campaign of education which shall build up a sentiment for a high mental and physical fitness as absolutely requisite qualifications for parenthood.
3. In all cases where the defect is unmistakable, segregation or asexualization.

The limitation of parenthood among the manifestly unfit or among border-line cases will by no means solve the problem of the prevention of epilepsy. Granted that an individual is brought into the world with a normally stable nervous system this stability is not indestructible. Syphilis, overwork, and undernutrition affect the child *in utero* and rob it of some of its stability. In labor, as we have indicated, delay, instruments, too great rapidity of delivery, asphyxiation do damage to the child which probably will show later in life if not at once. During infancy and childhood the normal characteristics of the organism are damaged by a wide array of unfavorable influences; faulty diet and nutrition are preëminent—children are well known to respond to indigestions with convulsions; the contagious diseases, both in the ordinary way through subjecting the patient to the stress of a severe intoxication or through direct attacks on the brain or meninges; lastly, the “bringing-up” of such children under conditions of slack discipline, nagging, and indulgence nowise fosters the important powers of inhibition; the whole organism becomes undisciplined,

and it is not to be wondered at that the child becomes moderately neuropathic and is prepared to transmit this taint to a future generation. It has been advocated that in addition to a proper diet and discipline such children should receive some mild sedative, as a bromide preparation, to assist in overcoming the instability which especially manifests itself through the nervous system.

Summary.—We have in the disease called epilepsy a syndrome arising from a number of diverse causes, which probably do not act singly in any one case but rather produce the disease by the sum of their activities. We speak of the condition as one disease because approximately the same mechanism is acted on in all cases, so that the symptoms are similar in spite of the diverse pathology. The necessity for the individualization of treatment is thus pointed out and likewise the necessity of approaching the case from many different directions. It must also be recalled that while the central nervous system is the apparent site of the disease it is possible that the syndrome may arise by action on the nervous system by remote products or stimuli origin.

Treatment and prognosis are affected by the mental state of the patient, by the chronicity of the disease, and also by the social and financial situation of the patient.

Routine bromide medication is to be condemned and recognized at its true worth—namely, as a palliative. A regular and hygienic life, with special attention to great moderation in the total intake of food and extreme care and thoroughness in keeping the digestive tract free from accumulations of waste materials, are of primary importance and far exceed the bromides in value as a routine treatment. Bromides are of value in cases which prove refractory to treatment and in whom otherwise there would be an excessive number of seizures; a little bromide will sometimes keep the seizures in such cases within safe limits; large doses should never be given as routine. In status and serial seizures the stronger sedatives, hydrotherapy, elimination, etc., must be used.

Surgical intervention is by no means a panacea. The brain must be handled at operation with the greatest delicacy and should not be lightly exposed. Prophylactic operation in traumatic cases is better than curative intervention; and in any surgical procedure the condition to be corrected must be balanced against the possibility of injury and of scar formation offered by the operation.

Institutional treatment for all epileptics is advocated as far as the public purse will permit. Recent cases are to be hospitalized; chronic cases provided with homes and work for the rest of their days; non-productive idiots and imbeciles housed on a custodial scale. The advantage of the institutional treatment over treatment at home lies in the better control of the case.

Lastly, prophylaxis by legal means as far as possible—segregation, operation, limitation of marriage; but above all by the education of the public to the dangers of matings between the unfit.

CHAPTER VII

THE TREATMENT OF THE MENINGITIDES

By WILLIAM J. M. A. MALONEY, M.D.

INTRODUCTION

THE treatment of inflammations of the meninges is governed by the same fundamental principles as direct rational therapy in all morbid conditions.

The meninges are serous membranes, similar to the peritoneum and the pleura, and may inflame as these by the action of bacterial or chemical irritants. Any specific attack, such as that of the tubercle bacillus, produces essentially the same morbid changes in the peritoneum as in the pericardium, and in the pericardium as in the meninges.

After the causes of a meningitis have been found, and appropriate remedial measures have acted, permanent changes in the tissue, due to the chemical action of the disease toxin and to the physical effect of the inflammatory exudate, may remain, and may render complete restoration of the function impossible.

The only perfect treatment for the meningitides is, therefore, prevention. In default of adequate preventive measures, the success of treatment depends largely upon early recognition that the symptoms arise from inflammation in the meninges, and upon accurate diagnosis of the nature of that inflammation.

In serous membranes, other than the meninges, the inflammatory processes are easy to detect. We feel and hear the friction of moving fibrin-clad surfaces; we see the distention produced by a fluid exudate; we discover the dulness of an exudate in a region which, normally, is resonant; and we see the impairment or abnormality of movement engendered by effusion or by adhesions. To guide us to the recognition of a meningitis, none of these signs is available. The meninges are concealed within the bony walls of the cranium and of the vertebral column. An inflammatory process in the meninges is impalpable, invisible, and inaudible. But, fortunately, there are other signs by which we may recognize its presence.

The meninges envelop the cerebrum, cerebellum, and spinal cord. The nerves which run to and from the central nervous system must pierce this meningeal envelope. Inflammation in the meninges may implicate a nerve in transit. Objective evidence of the inflammatory process, therefore, may be revealed in signs of irritation of the meningeal envelope and of the peripheral nerves. These irritative phenomena consist essentially of headache, pain, tenderness, and muscular

spasm. Pain may radiate throughout the distribution of the peripheral nerves; tenderness may induce a terror of movement; and muscular **spasm** may produce squint, rigidity of the neck, head retraction, **opisthotonos**, scaphoid abdomen, flexor or extensor spasms of the limbs, **retention** of urine, and constipation. If the irritation be sufficiently **intense** or prolonged, muscular palsies may ultimately result. As the **distribution** and intensity of the sensory and motor symptoms depend upon the site and the extent of the meningitis a study of the symptoms enables us to reach a fairly accurate topographical diagnosis.

In addition to the nerve roots the enveloped cerebrospinal axis may be implicated. The exudate from the meningeal inflammation, as it is confined within an inelastic bony cavity, will, if sufficient in amount, compress sensibly the underlying brain or cord. Such compression may directly interfere with the function of the nerve fibers and cells and may impede the venous and lymphatic circulation within the central nervous system. In consequence of these mechanical actions, edema and malnutrition occur in the nerve substance and cause disturbances of sensory and motor functions. The inflammatory process may itself invade the underlying nervous tissues, and to the meningitis add a myelitis or an encephalitis, or both.

The prominence of these irritative and pressure phenomena depends somewhat upon the intensity of the inflammation. Sometimes in a chronic inflammation an effusion gathers so gradually as to obscure the inflammatory nature of the process. The exudate, indeed, may be so circumscribed by adhesions as to produce pressure signs which suggest persuasively the presence of a tumor and the advisability of operative interference.

Diagnosis.—The first step in the treatment of meningitis—the diagnosis of the condition—is therefore difficult. Excepting the pain due to the pressure of the exudate upon the inflamed meningeal nerves, all the signs at our disposal arise from implication of structures other than the meninges—the underlying brain or cord or the penetrating nerves.

How can we be certain of the nature of the condition? The more acute the process the more urgent is the need to identify it. Unfortunately the greater also may then be the difficulty of diagnosis. A disease developing acutely with fever, malaise, irritability, vomiting, headache, rigidity of the neck, tenderness, head retraction, spasms and convulsions, with or without affection of consciousness, is probably a meningitis.

Differential Diagnosis.—*Hysteria.*—Headache, vomiting, convulsions, and rigidity may occur in hysteria, and such cases are not infrequently mistaken for meningitis. The absence of general toxic symptoms precludes an acute meningitic affection. To diagnose hysteria from tuberculous or serous meningitis is difficult. Heroic treatment by cold douches may arouse not only a hysteric but also a case of tuberculous meningitis. The symptoms in tuberculous meningitis sometimes by their daily variations suggest a functional disorder; so care is necessary.

The hysteric has no fever. Stigmas such as hemianesthesia, stocking, glove, or hand areas of anesthesia may happily aid in the differentiation. In hysteria the pupils are equal and react normally, objective signs of organic disease are absent, and the visual field may show characteristic contraction. Examination of the cerebrospinal fluid is of value in the diagnosis of such cases. The fluid is, of course, normal. But the diagnostic is inconsiderable compared with the therapeutic value of the puncture. If the little operation be performed with all due circumstance the cure of this particular group of hysterical symptoms is not only speedy but lasting. Rarely a second therapeutic puncture is necessary.

Uremic States.—The headache, drowsiness, irritability, vomiting, convulsions, and nerve palsies which may occur in uremic states may simulate meningitis. The urinary examination may not be very helpful for albumin, and casts occur also in the acute toxic conditions which can cause meningitis. Confusion arises chiefly in regard to the differentiation of uremia from cerebrospinal meningitis; but in uremia the fever is usually negligible, herpes is absent, and hyperesthesia does not exist. The treatment of the nervous phenomena in this state is, of course, that of the uremia—bleeding, saline infusions, hot packs, avoidance of morphine, chloroform, and other toxic symptomatic remedies, etc.

Pneumonia, exanthematous disease, and acute intestinal conditions have all to be excluded. In these the temperature usually rises more suddenly and attains a higher level; the pulse is faster; and the vomiting is more severe, more distressing, and more transient than in tuberculous meningitis.

Typhoid Fever.—It is often difficult to differentiate meningitis from typhoid fever. The meningitic is more irritable than the enteric patient. Stocker taught that if the bed-clothes be pulled down a typhoid case ignores it, but a meningitic case resents it and pulls the clothes up again. Jenner observed that when delirium appeared, complaining about the headache ceased if the case were typhoid, continued if meningitis.

Change in temperament appears in nearly all invalid states in neurotic children and adults. The diagnostic value of irritability or of drowsiness is therefore slight. Perhaps when the physician is first consulted only one of the enumerated signs suggestive of meningeal irritation is present in addition to the malaise, headache, and fever which are common to most toxic states. This initial sign is not constant in any form of meningitis. Thus in the posterior basic form in which cerebrospinal meningitis may appear sporadically in children, Lees and Barlow analyzed the initial signs as follows:

First signs—Vomiting	33
Convulsion	29
Head retraction	24
Screaming or irritability	9
Sleepiness or languor	7

102 cases

Infectious Diseases.—Convulsions and vomiting usher in many infectious diseases of childhood, for the stability of a child's motor and mental mechanisms is slight. More characteristic of meningitis is evidence of muscular spasm which usually appears in the first days. The earliest spasm occurs in the neck muscles: it produces head retraction, which although not pathognomonic is yet very suggestive of meningitis. Increased tonicity of the flexors of the leg when the thigh is flexed to a right angle with the abdomen (Kernig's sign) is so frequently present in meningitis as to be almost constant. Head retraction and Kernig's sign are presumptive evidence of the presence of meningitis.

When required to treat an inflammation of a serous membrane, say of the knee-joint, a physician's first endeavor is to identify the offending irritant so as to apply appropriate remedies. The salicylates which so powerfully aid in rheumatic are invoked in vain in tuberculous and are impotent in traumatic serositis. Treatment avails little unless it be directed immediately against the source of the evil. To establish the cause is a no less urgent and essential preliminary to the effective treatment of a meningitis than of any other serositis.

PALLIATIVE MEASURES

A conclusively established diagnosis requires time; but treatment may rarely halt until the nature and cause of the patient's ailment be discovered. What can be done in the interim to arrest the progress of the disease? If a case presents meningeal symptoms during an epidemic of cerebrospinal meningitis or of acute poliomyelitis the appropriate vaccine treatment should be employed without delay. Meningeal symptoms arising in an epidemic of meningitis are more likely to be due to the same cause as the epidemic than to any other cause. No harm will be done by the treatment, but incalculable harm may accrue from delay. Hence, in a case which may possibly be cerebrospinal meningitis there should be no hesitation in administering at the earliest moment an intraspinal injection of a meningococcic antitoxin. We shall soon have at our disposal an antitoxin for acute poliomyelitis also, which under analogous circumstances we may use. These are practically the only measures of specific treatment which we can employ upon a merely tentative diagnosis.

During this period of inevitable uncertainty the urgency of the symptoms may demand measures not directed against the elusive essential process.

Medicinal Treatment.—Morphine.—Morphine may be needed for the intense headache and for the restlessness, tenderness, pain, rigidity, and spasm. Its use in children cannot be too severely deprecated. It is difficult to control; alarming idiosyncrasies may be revealed even toward minute doses of the tincture of opium. Moreover, except to narcotize, little useful action is exerted by morphine. It relieves pain but it masks symptoms. The temptation to use morphine is powerful.

In the treatment of older children and adults it is seldom resistible. One may give to these $\frac{1}{8}$, $\frac{1}{4}$, or $\frac{1}{2}$ grain doses without much danger; even $\frac{1}{2}$ grain may be required to calm. But in infants below the age of one year the use of morphine should be banned, and in older children great caution should govern its employment.

Aspirin.—Aspirin and other coal-tar products such as phenacetin, acetanilide, and phenazone are used vaguely as antipyretics, analgesics, and hypnotics. In some cases they ease the patient by mitigating pain. The rheumatic diplococcus may cause an inflammation of the meninges, as of any other serous membrane, in which aspirin may be of service.

Urotropin.—The drug which, above all others, is useful in this stage of meningitis is urotropin. Crowe has shown that if urotropin be administered by mouth it can be detected within an hour in the cerebrospinal fluid. By heat, by the action of dilute acids or alkalies, urotropin can be converted into formaldehyde. The decomposition product in the body is probably formaldehyde or certainly a very closely allied substance which exercises a markedly retarding influence upon bacterial action. By inoculating the subarachnoid space of animals with streptococci of exalted virulence, Crowe has demonstrated that the attenuated solution of formaldehyde, which bathes the meninges when urotropin is administered by mouth, exerts an inhibitory influence upon the propagation of the organisms. Although the drug did not produce immunity to the infection, yet the progress of the streptococcal meningitis was slower and death was longer delayed in animals which received urotropin than in those which did not. Among those which received urotropin, the rapidity with which the inflammatory process spread could be markedly slowed by increasing the dose.

This salutary effect of urotropin has been repeatedly demonstrated. Obviously, the drug should be given at the moment any meningeal symptoms arise. Indeed, in persons exposed to infection in epidemics, prophylactic doses of urotropin are strongly to be recommended. By its aid a possible meningitis may be avoided, an incipient meningitis aborted, and an existing meningitis mitigated in virulence. Urotropin is not, however, always well tolerated by the gastric mucosa. It may be combined with bismuth. Frequently vesical tenesmus follows its use. Nicolaier reports a case where 90 grains per day caused blood and epithelial cells to appear in the urine.

It is quickly excreted and should therefore be given in frequently repeated doses in order to maintain its adequate concentration within the cerebrospinal system. As a rule the drug is well borne. The average dose for an adult is 5 to 10 grains every four hours. The frequency of administration may be increased until the first indication of intolerance be produced. Only thus can the full physiological effect be assured. In absence of definite knowledge of the nature of the exciting cause of a meningitis, urotropin is the most rational therapeutic agent which a physician can employ in the initial stage.

Mercury.—The headache and irritability may be relieved by purging. Mercury is probably the best cathartic. To infants one grain of gray

powder may be given thrice daily, or one dram of mercury ointment (*ung. hydrargyri*), or of white precipitate ointment may daily be spread on a binder which encircles the abdomen. Just as intolerant as children are of morphine so are they tolerant of mercury. The action of this drug partly consists in diminishing the absorption of toxic matter from the bowel by mechanically emptying and purifying the intestinal tract. Mercury is especially valuable when the presence of much indican in the urine shows the existence of excessive intestinal putrefaction. Mild purging, besides removing possible sources of toxemia, depletes the capillaries and lymph spaces of the intestines of fluid; the fluid content of the whole circulation is diminished; blood pressure falls; and thus the tension within the spinal and cranial cavities is lowered. Some of the drug is absorbed as a soluble albuminate of mercury, and is conveyed through the blood to the cerebrospinal axis. There the albuminate of mercury combines with the protoplasm of the meninges. The new protoplasm thus formed, by virtue of its mercury ions, seems to acquire an enhanced resistance to organismal invasion and an increased power to absorb and to dissolve the products of the inflammatory process. Hence, Gowers and the English school of neurologists strongly praise the efficacy of mercury in inflammatory processes in the meninges.

Bromides and Other Sedatives.—The patient may be so hyperesthetic that any stimulus troubles him; noise jars him, light vexes his eyes, and a touch induces reflex spasms. He may be as if he were suffering from strychnine poisoning. He may be unsoothable in his moaning and complaining; and if he be approached he may scream from fear of being touched. The elimination of all external stimuli, the quieting and darkening of the room, will comfort him. Bromides will diminish this reflex irritability. Their sedative effect may be reinforced by chloral. If vomiting be severe or if convulsions occur, chloral and bromide may not be administrable by way of the mouth, and may need to be introduced into the rectum, either in a suppository or in a small clystrum of bland medium. But chloral is a treacherous remedy, and surprising degrees of collapse not rarely follow its use.

Chloroform.—Chloroform inhalation is recommended by some as a sedative for convulsions, but it is even more dangerous than morphine.

Ice.—An ice-bag applied to the head may greatly alleviate headache. It is unnecessary to shave the head before applying the ice-bag. The ice may be crushed to render the bag colder and more comfortable. Salt may be added to further lower its temperature. If the patient be intolerant of the weight the ice-bag may be either suspended above or laid alongside the head.

Leeches.—The application of leeches over the mastoid region may relieve intracranial congestion and thus alleviate headache. Blisters behind the ears and pustulants, such as tartrated antimony ointment to the shaven scalp, are distressing to the patient and of little purpose.

Hydrotherapy.—The best means to ease the pain, to lessen the tension, and to tranquillize the irritability of the muscles is bathing.

Prolonged immersion is necessary. The calming influence of the bath is remarkable. Hyperpyrexia disappears, excitement abates, and sleep supervenes. If in a child the cerebral symptoms are associated with hyperpyrexia, awakening from the bath-induced sleep may be peaceful, but the temperature soon may mount again, twitching recommence, and a second bath be indicated. The relative merits of hot and cold baths for this purpose is a subject of controversy. Even the use of ice is advocated. Generally, however, a temperature between 65° and 85° F. is employed. The mode of action of the bath is complicated. The skin capillaries are as a great superficial sheet of blood. In a warm bath, as they are equally dilated, the depth and extent of the cutaneous blood pool is increased, the internal organs are depleted, and the heart, receiving less endocardial stimulation, slows restfully. The ducts of the myriads of skin glands are washed clean; the increased cutaneous blood supply stimulates the glands to free action; much fluid is eliminated; toxic matters are possibly excreted, and the blood improved. The diminution of pressure by the submersion of the thorax and trunk facilitates respiration and tends to make breathing slower, deeper, and more efficient. In addition to enhancing the circulatory, excretory, and respiratory functions, bathing reduces the temperature and thus mitigates the extravagant catabolism induced by the circulating toxins. The skin surface is the origin of numberless sensory nerves; these convey such a concerted volume of benign stimuli from the warmth and comfort of the bath that the irritable reflex arcs are tranquillized and the exhausted patient falls asleep. We possess no safer, no surer, and no speedier palliative for mental and for motor excitement than the bath.

If hyperesthesia be present, and bodily tenderness excessive, the manipulations involved in the transference from bed to bath and from bath to bed may be a source of anguish to the patient. He should be left upon the bed sheet undisturbed and thus lifted to and from the bath. If to lift him is impracticable, wet packs should be substituted for immersion. For a wet pack, a sheet wet with water of a temperature of about 65° F. is used. One-half of the sheet is rolled up; the roll is passed under the patient so that he lies upon the unrolled portion; the rolled part then is unwound to envelop him. The pack is removed after about an hour and the patient is wrapped in warm blankets.

LUMBAR PUNCTURE

As in the pleural, pericardial, peritoneal, and other serous cavities, so in the subarachnoid space, an increase of fluid may result from an inflammatory process. The pressure of this fluid exudate may cause many of the urgent symptoms. Hence, to relieve this pressure by lumbar puncture is no less efficacious than baths. Lumbar puncture is a procedure which is more urgently indicated in a meningitic than in any other condition, not only because of its immediate and

sedative effect, but also because of its great value in diagnosis. In lumbar puncture the intracranial tension is directly reduced by withdrawal of cerebrospinal fluid. As the amount withdrawn is under control the reduction of pressure can be regulated.

To understand the *rationale* of lumbar puncture we must consider the anatomy of the meninges.

Anatomy of Meninges.—There are three membranes or meninges covering the cerebrospinal axis—the dura mater, the arachnoid, and the pia mater. The dura, the pachymeninx, is the outermost investment. In the cranium it forms the periosteal lining of the bones and partitions the right from the left cerebral hemisphere, the right from the left cerebellar hemisphere and the cerebrum from the cerebellum. In the spinal cord it loosely envelops the arachnoid. The vertebræ have a periosteum of separate origin. The cleft between the dura and the arachnoid is known as the subdural space.

The pia mater or innermost membrane forms the immediate covering of the brain and cord, is closely applied to the outer surfaces of the cerebrospinal axis, and is invaginated into all the fissures. It is a highly vascular membrane; its inner surface is closely bound to the brain substance by the numerous small arteries and veins which pass between them.

Between the dura and pia lies the arachnoid. So intimate is the relation of the pia to the arachnoid that these are regarded as one membrane—the pia-arachnoid or leptomeninx. But between the arachnoid and the pia is a space, the subarachnoid space. Over the brain this space in some parts is obliterated, in others is expanded into large reservoirs or cisternæ. Around the spinal cord the space is very evident. The arachnoid is continued downward beyond the termination of the spinal cord as a tubular investment to the cauda equina. A large subarachnoid space is here formed, which may be called the caudal cistern. The subarachnoid space contains the cerebrospinal fluid.

Cerebrospinal Fluid.—Distribution and Circulation.—The cerebrospinal fluid, throughout the subarachnoid space, forms a more or less continuous fluid envelope. The fluid does not remain stationary nor stagnant. Its circulation has a definite direction and rate. One of Bier's pupils has shown that colored material, such as indigo, injected into the caudal cistern of animals appears at a cranial trephine opening within a number of minutes, which varies between ten and thirty according as the angle which the animal's spinal axis makes with the horizontal. This circulation is not a physical process. Control experiments show that in glass tubes of appropriate caliber containing fluid of a composition analogous to the cerebrospinal fluid, coloring matter takes incomparably longer to diffuse through a column of fluid the length of the spinal axis, placed at a similar angle.

The bilateral ligamentum denticulatum stretches from the arachnoid across the subarachnoid space to the pia so as more or less to partition the space into an anterior and posterior compartment. The septum posticum of Magendie and the arachnoid trabeculæ may form in the

cervical region a valvular arrangement which permits fluid to flow only in one direction.

Beneath the dura mater is a vast plexus of veins. When these veins fill the pressure in the vertebral canal is increased and cerebrospinal fluid is forced out of the canal into the subarachnoid space of the cranial cavity; when in inspiration and in the heart's diastole these veins empty, the pressure in the vertebral canal falls and the cerebrospinal fluid, partly by aspiration, partly perhaps by gravity, flows down the anterior compartment. Thus a cerebrospinal fluid circulation throughout the whole craniovertebral cavity is maintained.

An exudate, unless circumscribed by adhesions, increases the amount of cerebrospinal fluid and raises its pressure. The cellular and bacterial parts of the exudate become diffused throughout the subarachnoid space. Hence, by introducing a needle into the subarachnoid space, we may relieve intracranial tension and, from the withdrawn fluid, ascertain whether or not an inflammatory process exists in the meninges.

Technique of Lumbar Puncture.—For lumbar puncture a fine cannula or tubular needle, about 1 mm. in diameter and 4 inches long, with a sharp bevelled end, is used. A trocar or wire is inserted into the needle. The patient sits up, or lies on one side, with the body bent so that the lumbar region of the spine is arched toward the physician. Thorough antiseptic precautions are taken. The interspace between the lumbar spines immediately above or below a line joining the highest point of the iliac crests is selected. Quincke prefers the space between the third and fourth lumbar vertebræ and enters a few millimeters to one side of the middle line. The chosen area is frozen with ethyl chloride or anesthetized by infiltration with novocain. Often no local anesthetic is given. If opisthotonos or marked tenderness be present a general anesthetic is needed, especially in children. The needle is introduced. It should be grasped in the right hand; the penetrating end should be supported and steadied between the thumb and index finger of the left hand. Haste is undesirable. If the needle be unsupported and introduced rapidly it may impinge against a vertebra and snap. Considerable assurance is necessary in performing one's first lumbar puncture, for the needle point must penetrate from one to three inches before the subarachnoid space is reached—2 cm. in the case of a child, 4 to 6 cm. in the case of an adult (Quincke). When the point enters the space a lack of resistance to its progress is at once felt by the physician. The trocar or wire should then be withdrawn and the fluid allowed slowly to flow into the expectant sterile tube. Failure will be avoided if a free channel through the cannula be first insured and if the penetration occurs in the middle line. A slightly upward direction should be imposed upon the needle.

If it be desired to measure the pressure, practically no apparatus except capillary-bored glass tubing is necessary. Upon a piece of wood or card-board a paper millimeter scale should be fixed alongside the vertical glass tubing; this and a connecting glass T-piece, a few clamps, and some rubber tubing make an efficient manometer. The

horizontal limb of the T-piece and the vertical glass tubing are connected to the puncturing needle by means of short pieces of rubber tubing. A piece of rubber tubing is placed upon the vertical part of the T-piece and clamped. A clamp may be placed between the T-piece and the needle. The needle is introduced into the caudal cistern. The fluid flows out and the height to which it rises in the vertical glass tube is observed. Then the clamp may be taken off the vertical part of the T-piece and the fluid be collected in a test-tube for examination. It is neither necessary nor desirable first to fill the apparatus with saline.

The normal pressure of the cerebrospinal fluid is such that when the patient is recumbent the fluid issues in drops. At abnormal pressures the drops merge into a continuous stream. As estimated with Kronig's apparatus the normal pressure equals 125 mm. of water. A pressure of over 200 mm. (Boveril) or of over 150 mm. (Quincke) is abnormal.

If the patient be able to sit up the operation is greatly facilitated; but while the fluid flows the patient is safer in a recumbent posture.

Untoward Symptoms.—Most of the untoward symptoms are caused by the removal of too much fluid at one sitting. It is better to draw off several small amounts—say 3 fluidrams—(11 c.c.) at a time than to risk the possible consequences of one large tapping. In 1906 a child suffering from whooping cough and bronchopneumonia accompanied by convulsions was twice greatly calmed by the withdrawal of 2 or 3 drams (5 to 7 to 11.2 c.c.) of cerebrospinal fluid. At the third interference I allowed the fluid to continue to drain. After half an hour about 1½ ounces (40 c.c.) had collected, then the patient suddenly died.

In a youth suspected of rabies whom I "lumbar punctured" a paraplegia followed. In the course of three weeks rapid improvement occurred, but the patient was then still somewhat disabled. I was prevented from following his further course. The needle probably punctured a vein and the hemorrhage produced a pressure paraplegia.

How may these serious consequences be avoided?

The puncturing of a vein, although not of rare occurrence, seldom produces any evident consequences. It is an accident which cannot be prevented. The alarming collapse which sometimes follows lumbar puncture can be avoided if not more than 10 c.c. of cerebrospinal fluid are withdrawn at a sitting. How can one tell the moment at which the flow should be stopped? Quincke estimated the pressure before and after the removal of definite quantities of cerebrospinal fluid.

The following are the results in tabular form of his first cases:

Case.	Pressure of cerebrospinal fluid in mm. of water.			Amount of fluid withdrawn.
	Initial.	End.	Difference.	
1	210	85	125	19.0
2	250	110	140	18.0
3	200	110	90	18.0
4	155	120	35	8.0
5	165	70	95	18.0
6	130	45	85	1.5
7	200	70	130	25.0
8	300	150	150	15.0
9	280	170	110	10.0

Obviously, if a withdrawal of 1.5 c.c. can lower the pressure 85 mm. in one case and a withdrawal of 18 c.c. produces a fall of only 95 mm. in another case, the amount to draw off is a factor peculiar to the individual case. When a large fall in the pressure results from the evacuation of a small amount of fluid the cerebral subarachnoid space must be shut off from the spinal.

If the amount which one ought to withdraw varies with each case, what guide have we to determine it? The fall in the cerebrospinal fluid pressure affords us no index. A fall of 150 mm. during the operation may be as well tolerated as one of 35 mm.

Blood Pressure as a Guide.—The only reliable guide is the blood pressure. Horsley has long recognized and strongly insisted upon the necessity of carefully observing the blood pressure during intracranial operations. Marked lowering of the blood pressure is the only danger signal. Throughout the flow of the fluid the blood pressure should be carefully watched. If it falls considerably the withdrawal of the fluid should be stopped at once and the patient made to lie down with the head at a lower level than the feet. In case of alarming collapse the restorative is ether, hypodermically injected. In low circulatory states considerable time must elapse before a drug can be absorbed and conveyed by the blood to the central nervous system. Hence, a stimulant such as strychnine which is minus local action has in extreme collapse very little worth. But a drug such as ether which acts reflexly owing to its severe local and immediate irritative properties is invaluable. When a response has been obtained, strychnine and saline infusions containing adrenalin or pituitary extract, should be given to prolong the stimulation until natural recovery ensues.

In children, especially, it is advisable to have in readiness a hypodermic syringe full of ether, and to observe carefully the blood pressure during the spinal tapping.

About a score of deaths have been attributed to the use of lumbar puncture.

Sometimes no undesirable phenomena follow. Often, however, headache, vomiting, lethargy, and dizziness result which may be minimized or obviated by allowing the patient to rest in bed for a few hours after the puncture.

Results.—By lumbar puncture excess of cerebrospinal fluid is removed. Abnormally high intracranial pressure is thus reduced. With the reduction of pressure all pressure symptoms diminish; convulsions and twitchings stop; consciousness returns; and headache and vomiting and dizziness abate. Edema of the optic disk, if present, decreases. Toxic matter—bacteria and their products—in the exudate are removed and an inrush of blood into the meningeal vessels and of actively bactericidal serum into the lymph spaces occurs.

Examination of Cerebrospinal Fluid.—But apart from the relief of symptoms, lumbar puncture yields in the cerebrospinal fluid a valuable index to the presence and nature of an inflammatory process.

The cryoscopic examination—the estimation of the freezing point

of the fluid—has not yet proved of definite value and probably will not until it is associated with the examination of the electrical resistance of the fluid. The variation in the normal amounts of the sugar and galactose of the fluid have also an uncertain significance.

Mayerhofer's method of using permanganate titration as an index to the organic content of the cerebrospinal fluid is said to yield reliable results.

Noguchi claims that the use of butyric acid as a precipitant affords a delicate quantitative test for proteins. Normal cerebrospinal fluid contains 2 to 5 parts per 1000 of proteins and yields with butyric acid an immediate opalescent turbidity. In inflammatory conditions the globulin content is increased and the maximum normal turbidity is replaced by a granular or flocculent precipitate, which appears early and separates as a sediment. Noguchi maintains that the more rapid and distinct the reaction the greater the amount of protein present. The amount of protein is dependent mainly upon the amount of exudate. The exudate varies in quantity according to the nature and to the intensity of the inflammation. Hence, from the globulin content valuable deductions may be drawn.

But Quincke has shown that often excessive exudation may occur and the globulin content be no higher than normal.

Normal cerebrospinal fluid does not clot. When cells are injured fibrin ferment or its precursors are liberated. In inflammatory processes such liberation occurs and in addition fibrinogen is added to the fluid of the exudate. Clotting may then result. Such clotting appears if the cerebrospinal fluid be allowed to stand. The nature and extent of the clotting in certain diseases is said to be characteristic. Thus, in cerebrospinal meningitis a clot forms which is diffused like a cobweb through the fluid; in tuberculous meningitis a clot forms around a central point from which fibrin filaments radiate. In acute poliomyelitis no clot forms. The presence and nature of the clotting may therefore aid in diagnosis.

After centrifugalizing 5 c.c. of normal cerebrospinal fluid, cautiously decanting the supernatant liquid and carefully collecting (by means of a capillary pipette), fixing, and staining the minute trace of sediment which may remain we may find a few leukocytes in the field. Widal states that usually no cells are present; but with a magnification of 400, 3 or 4 lymphocytes may sometimes be found in a field. In inflammatory conditions, owing to cell exudation, several hundred leukocytes may be present. Indeed, in 1907 on an unknown young man brought comatose to my hospital service I performed lumbar puncture and drew off about 4 c.c. of pus. The number and nature of the cells is of diagnostic and of prognostic importance. Thus, in cerebrospinal meningitis and in septic meningitis, polymorphonuclear leukocytes are present in such large numbers as to render the fluid turbid.

In tuberculous meningitis the number of lymphocytes varies from 45 to 300 per cm.; the average is 160. In poliomyelitis the lymphocytes range from 10 to 45, and average 20 per cm.; when meningeal

symptoms arise in other infectious diseases lymphocytes may number from 3 to 15 per cm. (Netter). A vast amount of research has been devoted to the cytology of the cerebrospinal fluid. Recent work has tended to show that not only the nature but also the virulence of the process, and the stage at which the examination is made, may radically modify the cytological picture. Thus, polymorphs may be found in acute tuberculous processes, and mononuclears may be present in chronic meningococcic and septic conditions. As a general rule, however, polymorphs indicate an acute septic process; and mononuclears—when more than 10 per cm. are present—indicate a chronic, usually a tuberculous inflammation.

MENINGISM

We have hitherto discussed merely the palliative treatment of an illness of acute onset with meningeal symptoms. But while urgent measures are being taken for temporary relief, every effort should be made to identify the developing and underlying disease. Uncertainty prevails chiefly when dealing with children. All the symptoms of meningitis may arise in children solely from toxemia. The condition is then known as "meningism" or "meningismus." This meningism may be quite indistinguishable from meningitis. It arises sometimes in the course of the acute summer diarrhea of infancy; less frequently in otitis, pneumonia, and influenza; occasionally in diphtheria; and rarely in other acute contagious diseases, such as scarlet fever, whooping cough, mumps, etc.

All the symptoms of meningitis may be present in such toxic meningismic states, yet if the essential disease abate they subside quickly, or if death supervene, no morbid changes are evident in the meninges.

In children presenting meningeal symptoms examination of the lungs may reveal consolidation, and prove the nervous phenomena merely to be accompaniments of pneumonia or diphtheria, or even a benign sore throat may evolve from all the initial disturbance; or a rash may quickly show that the convulsions and vomiting were merely the prelude to an attack of scarlet fever; or meteorism, positive Widal reaction, enlarged spleen, diarrhea, and the temperature curve may disclose that the apparent meningitis was but the commencement of typhoid fever; or the development of bronchitis or diarrhea may prove the cerebral symptoms and bodily tenderness to have arisen from influenza; or a dose of calomel may show that all the furor was due merely to dietary indiscretion or perhaps even to intestinal parasites. So great apparently is the delicacy of the meninges, especially in children, that these various intoxications readily induce a reaction intense enough to simulate a meningitis.

Similar symptoms arose in a case of pellagra under my care and disappeared abruptly after three days. In a boy who presented fever, irritability, drowsiness, head retraction, scaphoid abdomen, and spasm

of the limbs, but no leukocytosis in the cerebrospinal fluid, no macroscopic changes were present in the meninges, but rabbits which were inoculated with this brain developed rabies (Bain and Maloney).

Charpentier, Vallin, and others have published cases in which meningitic symptoms arose in infants who were suckled by wet-nurses addicted to alcohol. Budin narrates such a case in which, after several days of sleeplessness and restlessness, violent prolonged attacks, characterized by convulsive movements supervening upon a state of more or less constant rigidity, occurred in the nursling of an alcoholic mother. The little one was quickly cured when the maternal feeding was stopped.

Many a case suspected of meningitis abruptly gets well and is renamed meningismus. What appears clinically to be meningism sometimes proves post mortem to be meningitis. Meningism and meningitis may both arise from the same bacterial irritants. At the onset of what later proves to be a meningitis the same symptoms may arise as in a case of meningism. Clinically and etiologically there is no fundamental difference between the two conditions. Meningism seems merely a transient incipient stage of meningitis.

Identical symptom complexes must have the same morbid basis. But it is alleged that no evidence of inflammation exists in meningism; whereas in meningitis the cellular exudate, tissue destruction, and repair and organization of the inflammatory products are in some degree always demonstrable.

Hyperemia, edema, and increase of cerebrospinal fluid have frequently been found post mortem in meningism. An increase in the amount of cerebrospinal fluid is detectable in the great majority of such cases, but the number of cells is unchanged.

Meningism is invariably the result of a toxemia. Toxins act as irritants to the meninges just as they do to other serous membranes.

The initial stage of inflammation is characterized by vascular congestion and exudation of plasma. If the irritation be inadequate in intensity or transient in action the inflammatory reaction halts there and emigration of cells from the vessels and lymph spaces is inappreciable. Under such circumstances the vascular disturbances may be too trivial to injure permanently either the meningeal tissue or the nerve cells; and the serous exudate may be too small or too slowly effused to produce pressure damage. As this irritation diminishes the effusion is absorbed, the circulation resumes its ordinary state, the tissues return to their normal condition, and function proceeds unimpaired. In fact, the pathological changes necessary to explain the symptoms of meningism have then been enacted.

Meningism is merely the initial stage of meningitis—a meningitis which halts before the period of cell destruction. It is incipient meningitis which aborts either at or before the stage of cellular exudation and tissue change. As cases of meningism are rarely fatal, microscopic examinations of the tissues after meningismic states have hitherto been too few and too incomplete to enable us satisfactorily to deter-

mine their pathology. It is interesting here to remember the cases of tuberculous meningitis *sans lesion* described by French pathologists. Voisin's histological research upon the central nervous system of fatal cases of pneumonia in children proved that even when meningeal symptoms were not evident in the clinical course, inflammatory changes were usually demonstrable in the meninges.

Meningism is etiologically, clinically, and pathologically a meningitis in which the disease does not progress beyond the initial stage.

With the amelioration of the essential condition the meningeal irritation subsides and the symptoms disappear. The meningitic reaction to the toxemia of diarrheal states in infants rarely proceeds further than an increase in the cerebrospinal fluid. The prognosis of such states is the prognosis of the intestinal condition. Their treatment is that of the inflamed bowel—freedom from all food, except water, until the bowel is tranquillized; warm baths to combat the collapse and to quiet the convulsions, and subcutaneous administration of salines to dilute the circulating toxins and to replace fluids drained off in the diarrhea. Rarely colon bacilli are found in a purulent meningitis.

In acute infectious diseases where an antitoxin is available, as in diphtheria, it should be given. The ordinary dietetic, symptomatic, and expectant treatment of the acute infectious disease should be followed.

Meningism and Serous Meningitis.—Quincke has described many cases of what he calls acute serous meningitis. This meningitis he considers to be either non-organismal or caused by organisms so few in number or so low in vitality that only serous and not cellular exudation results. The following is one of Quincke's reports and in its general features is typical of all.

Case 4.—Martha L., aged nine years. For several weeks somewhat deaf. On December 1 became acutely ill: excited, confused, vomiting, temperature 41° C. Subsequently only conscious at intervals.

December 4. Admitted to hospital. Temperature between 40° and 41° C.; somewhat stuporous; headache; neck very stiff and very tender. Paralysis of the left sixth; optic disk slightly blurred; veins distended; pneumonia of right lower lobe.

December 5. Lumbar puncture. Pressure 350 mm., 7 c.c. withdrawn. Pressure fell to 200 mm. Cerebrospinal fluid clear.

December 6. Pneumonic consolidation more marked. Coughing.

December 7. Temperature fell by crisis in the night. Head symptoms less.

December 8. Discharge of pus from both ears. Large perforation detectable in right; small in left tympanum. Staphylococci in the pus. Rapid resolution of the pneumonia.

December 21. Discharged cured, "with cicatrized tympani."

This case is nothing more nor less than a case of meningism appearing in a child in the course of an intoxication from a double infection. With this case, in which the origin of the toxemia is evident,

Quincke reports numerous others in which from some unknown source **auto-intoxication** arose periodically—sometimes in association with **the menses**—and produced transient meningeal symptoms. He and others describe cases arising from excitement, exhaustion, trauma, and infection.

Serous meningitis may arise, just as meningism, from infection by the pneumococcus, streptococcus, staphylococcus, *B. tuberculosis* (Lyonnert's four cases), *B. colon communis*, *B. typhosus*, the microorganism of rabies (Bain and Maloney), and other organisms. Probably when bacteriological and chemical methods are improved, investigation will show all of these cases to be due to microbic or autogenous irritants. Every organism already identified with serous meningitis is capable of producing a purulent meningitis. In serous meningitis these organisms are either less virulent or less numerous than in purulent meningitis. Hence, serous meningitis differs from graver types of meningeal inflammation, chiefly in the attenuated dose of the exciting irritants.

There seems to be no difference between meningism and acute serous meningitis. Each is merely an immature form of meningitis. When such an abortive meningitis occurs in an infant the name meningismus is invoked; when in an adult, acute serous meningitis. The essential distinction between meningism and acute serous meningitis is the patient's age.

Under the appropriate treatment for pneumonia, typhoid, otitis or other existing primary diseases the meningism or the acute serous meningitis may pass off entirely. All the measures which have been previously outlined for the tranquillization of patients with meningeal symptoms have here their application. But the most important is lumbar puncture. Doubt may exist as to the efficacy of lumbar puncture in the treatment of tuberculous, cerebrospinal, or other forms of meningitis, but no doubt can be entertained regarding its value in these abortive cases. Many or perhaps most of the symptoms in meningism and acute serous meningitis are due to high intercranial tension. This high tension is caused by the increase in the amount of the cerebrospinal fluid. Rarely meningismic symptoms appear when no such fluid increase is detectable. Apparently a præexudative stage then exists, a stage in which no reaction has taken place. In the vast majority of cases, however, increased pressure is present. The therapeutic value of lumbar puncture is then marvellous. Headache abates, rigidity diminishes, convulsions and twitchings cease; often the lumbar puncture seems to cure the disease as if by crisis.

The other remedy indicated is urotropin. By its aid we can procure that only a meningismus or a serous meningitis evolves, and that the meningeal inflammation, indeed, aborts. Baths, purging, sedatives, and the other symptomatic measures already indicated are also useful.

Toxins alone produce a fully developed meningitis. In fact, Cormand-Delille produced caseous tuberculous meningitis in animals by introducing the toxic products of the tubercle bacillus into the subarachnoid space. Laignel Lavastine demonstrated changes in the cerebral cortex

in general tuberculosis, in which no organismal invasion of the central nervous system could be detected. Voisin's work on the occurrence of cerebral changes in pneumonia may here be mentioned again (*vide* p. 288).

Although the initial stage of a meningitis is the same morbid process as in meningism or acute serous meningitis, the fact that certain organisms or their toxins rarely produce a meningitis so severe as to lead to cellular exudation or to more than transient affection of the central nervous system justifies the retention of the terms meningism and acute serous meningitis.

CEREBROSPINAL MENINGITIS

Cerebrospinal meningitis is infective and contagious. Its infectivity is slight; in hospitals, infection practically does not occur; in a family, as a rule, only a single case occurs, and cases are on record of a sound child scathlessly sharing the bed of an affected child. One of the main means of spread of the malady is by disease carriers—persons who without being ill themselves transmit the organism to others. Scarcely less dangerous are the mild abortive or ambulatory types of the disease. The disease affects chiefly children and young adults. The principal path of entrance of the organism is the upper air passages. Upon these few facts the prophylactic treatment is based.

Prophylactic Treatment.—Prophylactic treatment, however, requires the concurrence of the masses. The interest of the populace has scarcely dawned; concurrence is half-hearted or lacking; indeed, active opposition prevails.

Until we can educate public intelligence to be tolerant to the restrictions necessary for the preservation of the health of the community, prophylactic measures against meningitis must share the fate of the preventives against smallpox, malaria, typhoid fever, tuberculosis, syphilis, and other plagues. Only in the bitterness of visitation, when meningitis devastates a district—as it lately did, in Texas—so that social, business, and political influences are dwarfed by its appalling presence, is concerted action taken by the community to eradicate and to prevent it.

In times of epidemics the congregation of children should be stopped by closing schools, playgrounds, and other resorts of child society. Cases should be rigorously segregated. Segregation is thought to be impractical because of the existence of healthy carriers of the organisms and of ambulatory forms of the disease. But, as completely as possible, every infective person should be isolated. Every infected person taken out of the community removes a centre for the creation not only of cases of the disease but also of disease carriers. These disseminators of the disease need not be treated as sick, but they must be eliminated from the society of the healthy until they become non-infective and harmless. To trace them is often difficult. Compulsory notification

of cerebrospinal meningitis should be enforced under heavy penalties. The origin of every infection should be sought without ceasing. The ticketing of affected houses and their isolation is desirable. Examination of the nasopharyngeal mucus of all inmates and habitués of affected dwellings should be made. "It is not rare," says Wassermann, "to find for one patient sixty healthy persons in whose nasal mucus meningococci can be demonstrated." The frequent syringing of the nasopharynx with a 1 per cent. solution of hydrogen peroxide or with a 1 in 200 solution of formaldehyde will sterilize these infected nasopharyngeal cavities. The administration of urotropin in 10-grain doses twice daily to each exposed person tends to protect them against the disease. The malady can be confined to the house implicated and to the initial patient. Still more efficacious as a prophylactic measure is the injection of meningococcic antitoxin. The antitoxin is injected subcutaneously. A single dose of 10 c.c. is given. Its results seem to be very satisfactory.

Diagnosis.—As regards the actual treatment of the condition the first essential is to be sure of the diagnosis. In times of epidemic delay is dangerous. A child showing meningeal symptoms should at once have the benefit of an injection of an antimeningococcic serum. In sporadic cases a physician is more conservative. The possibility of meningitis being due to the meningococcus exists always, but the possibility of the occurrence of a sporadic case of cerebrospinal meningitis, in an otherwise free neighborhood, is less than that of tuberculous or septic meningitis. As the injection of antimeningococcic serum involves no danger, for in cases of meningitis which are not due to meningococci it is merely useless, and as in cerebrospinal meningitis its efficacy depends upon its early administration, its routine employment at the first suspicion of meningitis is strongly commended. The absence of detectable tuberculosis in the child; the lack of meteorism, of diarrhea, and of Widal reaction; the severity of the initial vomiting; the sudden high temperature, not rarely above 40° C.; the presence of herpes labialis, the early rigidity of the neck and back, with Kernig's sign, all arouse suspicion of cerebrospinal meningitis. Lumbar puncture usually establishes the diagnosis. The fluid is at high pressure and is turbid. It is characteristically coagulable. The sediment deposited is considerable, but the protein content of the supernatant fluid is low. A great number of polymorphonuclear leukocytes are present, many of which enclose the *Diplococcus intracellularis meningitidis* of Weichselbaum. The presence of this organism may be demonstrated in the mucus of the nose and throat also.

Antitoxin Treatment.—If the presence of cerebrospinal meningitis be certain, antitoxin should at once be given. A great number of antitoxins are available. All are prepared in the same way; all consist of the serum of an animal which has been actively immunized against the meningococcus by repeated injection of various strains of meningococci in progressively increasing doses. The initial injection consists of toxin or dead organisms; then living organisms are used of a virulence

which may gradually be exalted. Wassermann, Jochmann, Flexner, Dopter, and others have prepared such antitoxins and demonstrated their value. Formerly subcutaneous injections were employed. Failure materially to influence the course of the disease often resulted. But with the adoption of intraspinal administration the reliability and efficacy of this mode of treatment have placed it among the greatest achievements of modern therapy. This specific treatment should be given at the earliest possible moment. Indeed, I have already indicated that its prophylactic administration in the healthy and its use in the suspiciously ill, are our two most powerful weapons in combating epidemics.

Results.—The results are little less than marvellous. Often the temperature falls at once, the symptoms abate, and the disease ends as by crisis in a few hours. More usually the severity of the illness steadily diminishes, the vomiting ceases or becomes less frequent, profuse sweating occurs, the patient begins to take nourishment, and within twenty-four hours the alarming aspect of the case disappears. Complications are rarer and less dangerous, and the duration of the malady is greatly lessened under the action of the antitoxin.

Types of Reaction.—Levy distinguishes three types of reaction. In the first, after the initial injection, the temperature immediately falls but rises again; after the second injection the temperature subsides by steps to a normal level. Coincident with the lowering of the fever a progressive amelioration of the symptoms occurs.

In the second the fever tends to persist, but the headache, optic neuritis, and rigidity of the neck lessen continuously and alimentation becomes easy; the symptoms gradually disappear: cases of this type are liable to relapse.

In the third no marked results follow the injections, but the case mortality is lowered.

In the great majority of cases the immediate result of the injection is a lowering of temperature and a marked improvement in all subjective symptoms. No case is hopeless. Recovery after the disappearance of the skin, knee, and plantar reflexes, after incontinence, and even coma, is not uncommon.

Owing to the great reflex irritability it may be desirable before attempting the intraspinal injection to administer a sedative injection of morphine and to employ local anesthesia. The volume of cerebrospinal fluid allowed to flow should be greater than that of the serum injected. For example, 40 to 60 c.c. of fluid may be cautiously withdrawn and 20 c.c. of serum injected. The serum should first be heated to body temperature, but no higher. If pain occur during the injection it will be only momentary. After the injection the pelvis should be raised and the head lowered so that gravity may assist the circulation of the serum within the thecal canal.

Dosage.—Various doses are advocated. Wassermann and Liebler recommend that to children under one year, 10 c.c. should be given; to children over one year, 20 c.c.; to adults, 25 to 30 c.c. Jochmann

counsels that 20 c.c. be employed for those under fourteen years; 10 c.c. for those less than one year. In severe cases even larger doses are advisable.

Early Administration.—The great factor in the success of the treatment is the early administration of the serum. If the disease be recognized at the onset and the serum be given at once the further progress of the illness may be prevented. The injections should be repeated at short intervals of about eight to twenty-four hours. No more than two days should be allowed to intervene between each injection. When three or four days have been permitted to elapse, death from anaphylaxis has occasionally resulted. The injection should be continued until the cerebrospinal fluid is clear to the unaided eye and the headache, anorexia, vomiting, neck rigidity, optic neuritis, etc., are markedly ameliorated. Sometimes three or four injections are necessary before improvement is evident. Usually the total amount of serum given ranges from 40 to 90 c.c., but as much as 210 c.c. has been administered.

The action upon the individual case has already been dealt with. The effect upon the death rate is wonderful. Wassermann states that the mortality without serum treatment varies from 30 to 60 per cent. in different epidemics. In the epidemic in the Rhenane province and Westphalia, which was reported by E. Levy, the death rate among those not treated by the specific serum was 78 to 80 per cent.; whereas among those treated with the serum it was only 12 to 15 per cent. Chatelet and Friand, as the result of their researches, decide that the average case mortality before the use of antimeningococcic serum was 30 to 40 per cent. They publish notes of an outbreak of 30 cases which occurred at the garrison at Verdun and which they treated with serum. Only one case ended fatally. Jochmann estimates the reduction of the case mortality by the serum treatment as 25 to 35 per cent. If large and repeated doses be given in the early stages of the disease the success of the serum treatment of cerebrospinal meningitis will rival that of the antitoxin treatment of diphtheria.

Sometimes the action of the serum is accompanied by marked albuminuria of a transient nature. The usual accidents of serum therapy, urticaria, erythemas, and joint effusions, may supervene. These usually are preventable by the administration of atropine or of calcium chloride. Death from anaphylaxis may occur. Vigot reports such a case and refers to others published by Hutinel and Darré, Sicard and Salin. In order to avoid this danger massive doses following fast upon one another should be given early in the disease. If uncertainty exists, for instance in the case of a relapse, as to the presence of anaphylaxis, instead of injecting the serum into the spinal canal, a tentative minute dose should be given per rectum or subcutaneously and its effect carefully observed before more radical treatment be attempted.

If the treatment be delayed until permanent damage has been produced the serum, although it can arrest and stamp out the inflammation, cannot remedy nerve destruction. Thus if an optic neuritis

be severe, although it may disappear under serum treatment, some degree of optic atrophy is sure to remain. Similarly, nerve deafness may result. But from the moment of administration of the serum inflammation of nerves halts and begins to retrogress.

Purulent eye, ear, and joint conditions, albuminuria and glycosuria may complicate the disease. Under serum therapy such complications are rare, of short duration, and of mild degree.

When after adequate and repeated exhibition of the serum the disease does not abate, then one should suspect the accuracy of the diagnosis. The inoculation of a guinea-pig may prove the meningitis to be tuberculous.

The other measures already described for the treatment of septic meningitis—the administration of urotropin, the repeated withdrawal of cerebrospinal fluid by lumbar puncture, careful feeding, periodic withdrawal of urine in cases of retention, etc., must reinforce the serum treatment. For details regarding their employment the reader is referred to pp. 278 and 280.

The treatment for cerebrospinal meningitis is antimeningococcic serum, which alone is specific, safe, reliable, and adequate.

MENINGITIC FORM OF ACUTE POLIOMYELITIS

As the most constant of all pathological lesions in acute poliomyelitis is a cellular infiltration of the pia, it is not surprising that this disease should sometimes convincingly resemble some form of meningitis. It is itself essentially a meningitis to which a myelitis or an encephalitis is added. Headache, vomiting, pain and rigidity, and spasm of the muscles, all the symptoms of meningitis, may occur in poliomyelitis. So persuasively may meningitis be simulated by poliomyelitis that few epidemics of poliomyelitis have been recorded in which the synchronous outbreak of meningitis has not been reported. Thus, Leegaard in his classic report of the Norwegian epidemic of acute poliomyelitis of 1903 reported synchronous cases which he thought were cerebrospinal meningitis. Netter in Paris and Caverly and MacPhail in America also reported that cerebrospinal meningitis and poliomyelitis may occur simultaneously. Indeed, a belief was formerly current that these two diseases are related.

Further experiences, especially in the recent appalling epidemics of poliomyelitis, have shown this belief to be unfounded. Now these cases of poliomyelitis have been segregated and are regarded as a separate group—the meningitic type of acute poliomyelitis. As this type differs from an ordinary meningitis only in the subsequent history of the ailment it is necessary to consider it here.

Symptoms.—The various symptoms already enumerated for meningitis may all be present in acute poliomyelitis. Almost any symptom may predominate; almost any possible combination of symptoms may be present. In some cases the signs of meningeal irritation, although alarming, are evanescent. In others the meningeal signs become more

and more evident until the malady ends fatally. In others, again, characteristic palsies appear after a few days and leave no doubt as to the diagnosis.

In the mildest cases only pain in the neck develops. Frequently, however, rigidity of the neck is also present, although it may be so slight as to be demonstrable only on bending the head forward. In more severe cases the head may be retracted, sometimes markedly. Wickmann states that head retraction may be present without muscular spasm being evident. Not seldom to these symptoms is added stiffness, with pain in the back; occasionally, orthotonos; more rarely, opisthotonos; sometimes tenderness of the spinous processes of the vertebræ may be present. Kernig's sign has been noted.

These signs of meningeal irritation are particularly evident in the initial stage of the disease, the stage in which the invasion of the pia occurs. Netter states that one-third of the cases of poliomyelitis begin with distinct symptoms of meningeal irritation.

Wickmann cites the two following cases as typical of the meningeal form of acute poliomyelitis:

The first developed convulsions, marked somnolence, stiffness of the neck, opisthotonos, hyperesthesia, persistent tonic spasm of the limbs, strabismus, inequality of the pupils, and retention, with subsequent incontinence of urine. All these symptoms disappeared within two weeks and left no trace.

The second, an adult, had fever, headache, vomiting, pain and stiffness in the neck, and tonic spasm in some of the muscles of the shoulders and arms. Later, cramps in the legs appeared; then opisthotonos; and finally, difficulty in speech and in swallowing developed. Death came three days after the illness began. Postmortem examination showed in the spinal cord the changes typical of acute poliomyelitis.

Diagnosis.—The diagnosis of the cause of the disease is often a matter of extreme difficulty. Mention has already been made of the fact that so similar clinically is the meningitis caused by the virus of acute poliomyelitis to that which arises from the meningococcus intracellularis that these diseases were formerly supposed to have some fundamental relation to one another, and reports of the prevalence of cerebrospinal meningitis during epidemics of acute poliomyelitis are common.

There is, of course, absolutely no connection between the two conditions. How are they so often confounded? How can they be differentiated?

When a crop of cases of meningitis springs up the physician's first thought is that there is an epidemic. The only epidemic form of meningitis which is generally known is cerebrospinal meningitis. Hence, the erroneous diagnosis is suggested. Even when the case is very carefully examined its real nature may not be positively ascertainable (cf. the four cases reported from the Karolinen Kinderspital, page 298).

If, after a short febrile period, flaccid paralysis develops a diagnosis

of poliomyelitis is justified. The differential diagnosis from similar conditions due to other infections, during the preparalytic stage, will be discussed in the section on acute poliomyelitis. Here we shall consider only the separation of this disease from cerebrospinal meningitis and tuberculous meningitis.

A relatively brusque onset, characteristic herpes labialis, early and marked rigidity of the neck, high temperature, psychic disturbances, prominence of the cerebral symptoms, all tend to a diagnosis of cerebrospinal meningitis.

An insidious onset, changes in temperament, relatively low degree of fever, photophobia, mild type of vomiting, with ordinary signs of meningeal irritation, among which headache, constipation, and scaphoid abdomen are often emphasized, are suggestive of tuberculous meningitis. The presence of tuberculosis elsewhere would practically confirm the diagnosis.

But certainty in diagnosis from merely clinical observation is difficult to attain. Only in the data obtained from lumbar puncture can we place implicit reliance. In all three conditions the fluid is at high pressure. In cerebrospinal meningitis it is, however, turbid, shows polymorphonuclear leukocytosis, and usually the intracellular meningococci can be detected.

Lumbar puncture in tuberculous as in the meningitis of acute poliomyelitis yields a clear fluid which is under increased pressure. The fluid in both shows lymphocytosis, the average in tuberculous being 160 lymphocytes per centimeter; in acute poliomyelitis, 20 per centimeter. The fluid coagulates, but in tuberculous meningitis the clot forms about a characteristic central thread, whereas in poliomyelitis it is diffuse and crumbling. As has already been shown, the resemblance between these two conditions may be very remarkable. If the presence of tubercle bacilli can be demonstrated the difficulty at once disappears. Upon the appearance of the characteristic flaccid paralysis, with diminution of tendon reflexes, atrophy and reaction or degeneration, no further doubt exists as to the diagnosis.

Treatment.—Prophylactic Treatment.—The labors of Wickmann have shown that acute poliomyelitis is an infectious disease, which is conveyed from person to person. Wickmann found also that the disease tended to occur in groups, to radiate from a common centre, and to diffuse along highways. He showed that healthy intermediaries—so-called carriers—were a fertile source of infection. Indeed, the conditions which prevail in acute poliomyelitis are precisely analogous to those in cerebrospinal meningitis and demand the same preventive measures (*vide* p. 290). But, whereas, in cerebrospinal meningitis we can demonstrate the presence of the causal organism in the nasal mucus (*vide* p. 291) of the disease carriers, in poliomyelitis we possess no such guide. Only the track of the disease indicates the identity of a disease carrier. Similarly, abortive cases can be diagnosticated only presumptively. Hence, effective isolation is impossible and prophylaxis is imperfect.

As the intestinal and nasal mucosæ and the salivary glands are excretory channels for the virus the destruction of their secretion is advisable. One per cent. solution of hydrogen peroxide will destroy the virus in the buccal and nasal cavities. The following powder has also been recommended for this purpose:

R—Menthol	1.2 grams
Salol	5.0 grams
Boric acid	20.0 grams

Specific Treatment.—Serum treatment in this disease is still in its infancy. It has been proved that monkeys which have recovered from a first attack resist successfully a second inoculation. Antibodies can be demonstrated in the blood of such immune monkeys and in that of children who have recovered from the disease. It has also been proved that even when the disease is so slight as to produce no characteristic clinical symptoms this immunity may be present. Every requisite, therefore, seems here to be present for the production of both active and passive immunization against the virus of acute poliomyelitis. Monkeys have been successfully immunized by a method analogous to the Pasteur inoculation for rabies. Hitherto immunization has practically not been attempted for man.

Symptomatic Treatment.—The measures already enumerated for the relief of the symptoms of meningeal irritation should be employed. Wet packs, urotropin, and especially lumbar puncture are of great service. (For further details see Treatment of Acute Poliomyelitis.)

TUBERCULOUS MENINGITIS

Prophylaxis.—The preventive treatment of tuberculosis of the meninges does not differ in any essential particular from that employed to prevent tuberculosis elsewhere. The enforcement of the laws of public and private hygiene; milk sterilization, fresh air, good food, and sunshine; the treatment of existing tuberculous conditions in other regions; the early evacuation of tuberculous abscesses, especially those which occur in the neighborhood of cranial or vertebral bones, are all safeguards which are self-evident.

The degree of an individual's resistance to any organism can be estimated. When a person has a normal resistance he is said to have a normal opsonic index with regard to that organism. The resistance of a person to tubercle bacilli can be ascertained easily. By the injection of tuberculin in adequately minute doses a low resistance can be exalted, a low opsonic index to the tubercle bacillus can be raised. As the technique is complicated and the danger of ignorance considerable this mode of therapy has so far had only a limited field.

Diagnosis.—The recognition of tuberculous meningitis is often extremely difficult. The absence of cerebrospinal meningitis in epidemic form, of herpes labialis, of polymorphonuclear leukocytosis

in the blood, and of severe vomiting is presumptive evidence **against** the presence of a meningococcal infection. The blood picture and the absence of pulmonary signs render a pneumococcal invasion **unlikely**. Constipation, absence of meteorism, negative Widal, and rigidity of the back or opisthotonos suggest that typhoid fever is **improbable**. An insidious onset, with irritability, change of temperament, **apathy** or somnolence, and headache is supposed to be a customary mode of onset, but nervous people are thus affected at the beginning of **most** toxic conditions. Photophobia occurs frequently in tuberculous **meningitis**; in the posterior basic form in which cerebrospinal meningitis may appear sporadically, retraction of the upper lid may be observed. Tubercles in the choroid may prove conclusively the nature of the affection. The difficulty of diagnosis could not better be illustrated than by four cases admitted to and reported from the Karolinen Kinder-spital. For a week or more the well-known prodromal symptoms of basic meningitis prevailed. Not only were there changes in disposition, tiredness, vomiting, nocturnal restlessness, screaming, occasional restlessness, and obstipation, but convulsions suddenly appeared associated with marked rigidity of the neck, irregularity of the pulse, Kernig's sign, transitory increase of the patellar reflexes, strabismus, general cutaneous hyperesthesia, vasomotor disturbances, and typical cerebral facies. The deception was still further enhanced, for lumbar puncture yielded a clear fluid which was under pressure, which formed a distinct, non-reticular fibrinous clot, and which, on cytological examination, showed only numerous lymphocytes. At length the noteworthy retrogression of the signs of irritation, and later of the cranial nerve symptoms; the complete disappearance of the fever; the more or less localized flaccid paralysis of limb and of abdominal muscles, together with the appearance in them of the reaction of degeneration; and recovery either complete or associated with permanent more or less diffuse flaccid paralysis and atrophy of the muscles, admitted of no doubt that these were cases of acute poliomyelitis.

The cutaneous reaction to tuberculin vaccination—the von Pirquet reaction—is inconstant and uncertain in its indications. The Calmette reaction to the introduction of tuberculin into the conjunctival sac has the same uncertainty, with additional danger.

In the presence, therefore, of a febrile disease presenting the symptoms of meningeal implication, which have already been described, if no signs of tuberculosis be present in other areas the diagnosis can only be tentative until the cerebrospinal fluid has been examined.

The fluid is clear. Usually it is excessive in amount and under considerable pressure.

The pressure, however, is not a specific feature of the infection. It varies tremendously and is usually higher in adults than in children. Of 64 cases of tuberculous meningitis, 23 per cent. showed a pressure of between 200 and 300 mm. of water; 23 per cent. between 400 and 500; 19 per cent. between 300 and 400; 17 per cent. between 500 and 600; 8 per cent. between 600 and 800; 6 per cent. between 100 and 200;

1.5 per cent. between 50 and 100; and 1 to 1.5 per cent. over 800 (Quincke).

Out of 44 cases, 12 showed over 2 parts per 1000 of albumin; 5 over 3; 3 over 4; 1 over 5; and 1 over 6. But occasionally even when the fluid is greatly increased in amount the albumin remains less than $\frac{1}{2}$ per 1000 (Quincke). Rarely no deviation from the normal, physical, and chemical characteristics is present. In 50 per cent. of cases the fluid coagulates. The clotting may occur when only $\frac{1}{2}$ per 1000 of albumin is present. The fibrin filaments throughout the fluid are connected with a characteristic central thread. Later the clot crumbles into a sediment. The sediment in this disease is less and the albumin of the supernatant fluid is greater than in cerebrospinal meningitis. But more characteristic are the contained cells. The cells are mostly lymphocytes. Many of them may show degenerative changes. The number present averages 160 per cm., but varies between 45 and 300. Certain cases of acute tuberculous infection of the meninges have, however, been described in which the polymorphs predominated. Forbes examined 80 cases of tuberculous meningitis, 70 of which he verified. He found excess of lymphocytes in 51; excess of polymorphs in 5; and equal proportion of lymphocytes and polymorphs in 4. Sometimes no increase in the cells is found in cases which are ultimately proved to be tuberculous. And occasionally in normal cerebrospinal fluid a few lymphocytes are present.

Tubercle bacilli may be demonstrable in the fluid. If a stained smear from the sediment deposited by centrifugalizing does not reveal bacilli they may be detected in 99 per cent. of cases (Bernstein) by incubating the fluid for forty-eight hours and then examining the clot. If no bacilli can then be found inoculation of a guinea-pig with the fluid will certainly demonstrate them if they are present.

The absence of indications of the presence of other organisms or of their products will help to substantiate the tuberculous origin of the process.

The sugar-reducing properties of the cerebrospinal fluid upon which some stress was formerly laid are now known to have very little diagnostic value.

In doubtful cases it is advisable to act upon the presumption that the disease is cerebrospinal meningitis and actively to treat it accordingly (*vide* p. 291). The antimeningococcic serum is innocuous, and less harm lies in its useless administration in a tuberculous condition than in unduly withholding it in an atypical case of cerebrospinal meningitis.

Treatment.—When the diagnosis of tuberculous meningitis is established what treatment may be employed? As regards life there is nothing in the morbid process fundamentally contrary to continued existence. Between 1894 and 1909, 20 cases of undoubted cure were reported (Martin). The disease under favorable circumstances, therefore, may spontaneously disappear. Complete restoration of function may even occur. Therefore, our efforts should be centred upon the

maintenance of the patient's strength and resistance. The patient must be fed adequately. Successfully to nourish a vomiting patient will tax the resources of the most ingenious physician. Food in small amounts frequently, in easily ingestible and assimilable form, is required. Fluid food, predigested, may be necessary. Especially in children, and in the semiconscious, feeding by means of the nasal or the stomach-tube is valuable. Before feeding the stomach should be washed free of any fermenting, irritating contents. The ease of feeding varies curiously from day to day. Some days the patient takes food if not with zest at least without resentment. Other days absolutely no interest can be elicited. Swallowing itself is sometimes easy, sometimes difficult, sometimes apparently impossible. Stomach feeding may be reinforced by nutrient enemas. Inunction with cod-liver oil is practised, but it is unpleasant and of doubtful value. Subcutaneous injections of sterile olive oil, of horse serum, and of normal saline have a certain value as adjuvants to other nutritive measures. Alimentation is the central problem in the treatment of tuberculous meningitis.

An attempt to raise the opsonic index may be made. Great caution is necessary. The opsonic index should first be ascertained. It is liable to fluctuate. When the resistance is increasing a "positive phase" is said to be present. When it is decreasing a "negative phase" exists. Injection of tuberculin should be made only in minute doses, and only when a positive phase is proved to be present. Otherwise the injection may cause a more or less localized process suddenly to disseminate widely.

Tuberculin.—Tuberculin is obtainable in many forms, but Koch's tuberculin (T. O.), tuberculin residue (T. R.), bouillon filtrate (B. F.) are chiefly used. T. R. contains the pulverized tubercle bacilli. It is therefore a vaccine and should immunize actively against the tubercle bacillus. The bouillon filtrate should immunize against the toxic products of the tubercle bacillus. Every precaution is taken to insure the sterility of the injection. Usually for this purpose small quantities of antiseptics are added.

One cannot proceed too cautiously with these injections, as I once sadly experienced. The administration should be repeated at intervals. When $\frac{1}{1000}$ gram is being given it may be repeated every third day. If a "reaction" results the treatment should be suspended and the dose diminished. Such a reaction may manifest itself in general symptoms, such as headache, malaise, and fever; or the signs of the tuberculous inflammation may be aggravated; or at the inoculation site an inflammatory reaction may occur.

Lumbar Puncture.—Marked success has been reported in the treatment of tuberculous meningitis by means of repeated lumbar puncture. By this procedure organisms and toxins are removed; the intracranial tension is lowered, vomiting, headache, and spasm are relieved; and an influx of bactericidal cerebrospinal fluid to the diseased area is promoted. As much as 70 to 90 c.c. may be withdrawn daily. At each puncture 10, 20, 30, or 40 c.c. may be allowed to flow off. The

pulse should be carefully watched. The patient should be kept recumbent. If much difficulty is experienced in inserting the needle the patient, if well enough, may be seated, but while the fluid flows he should be kept recumbent. A hypodermic syringe full of ether should always be kept ready to combat sudden collapse during this tapping of the thecal cavity (*vide* p. 284).

Von Bier's Treatment.—Von Bier's principle has, of course, been employed in this condition, and ingenious devices for its application have been constructed. Headache hyperesthesia, optic neuritis, and vomiting are not symptoms which invite an attempt to increase the congestion. I have no personal experiences of the method in tuberculous meningitis, but reports of its benefit are meagre and unconvincing.

Medicinal Treatment.—The administration of sodium iodide in massive doses, even as much as 600 grams per day, apparently coincided with several recoveries. Injection of iodoform emulsion, both into the sub-arachnoid space and subcutaneously, has been highly lauded. Mercury has its advocates. Few drugs in the pharmacopœia have not been tried. Cures from the use of each drug have resulted only when they were administered by their discoverers or their chosen apostles. In other hands, iodide, iodoform, etc., have been useless. Cures which ensue from methods so fastidious in their activity as only to be efficacious in the hands of their believers are accidental to and not consequent upon the use of the alleged cure. When a disease has so many "specific" remedies there is a high degree of probability that all are impotent. The therapy of every disease which is liable to spontaneous remission or to natural cure is wrapped in the nostrums of physicians who are more clamorous than critical regarding their practice. They "look upon their work and, behold, it is well done," invariably. Frederick Taylor writes, "undoubtedly tuberculous meningitis is in nearly all cases fatal; and the prognosis may be regarded as unfavorable in proportion to the certainty of the case being tuberculous." In many cases an opportunity arises for an extensive trial of several remedies. The chief value of these remedies lies in their power to reassure and to satisfy the parents and the physicians. If the disease happily abates it is a cause for thanksgiving, not a result of skill.

Surgical Treatment.—In this, as in most chronic diseases, surgery has been tirelessly practised. Laparotomy is so often beneficial in tuberculous peritonitis with effusion that it seems a rational inference that craniotomy would be of service in tuberculous meningitis. The tuberculous meninges, however, have been subjected unavailingly to every manipulation known to surgeons. Stiles reported two recoveries after trephining and draining. Isolated cases of improvement have been recorded by other surgeons. The after-history of the cases thus benefited is problematical. It has already been mentioned that this disease tends occasionally to spontaneous healing. The wonder is, not that a few successes have been reported, but that out of the multitude of surgical attempts more did not happily occur upon those who in any case would have recovered. To add to the low vitality of a case of tuberculous

meningitis the profound shock inseparable from intracranial procedures usually suffices at once to end the patient's life.

Adhesions may result from the organization of the inflammatory products. Thus, areas are circumscribed within which serous exudates accumulate and produce focal pressure symptoms. Often such cases are operated upon with intent to remove a suspected tumor. In skilled hands the patient derives only benefit from the error; with the release of the fluid the pressure symptoms disappear, for the focus of pressure—the serous exudate circumscribed by adhesions—is tapped. Those cases in which nothing at all is found at the operation and which subsequently improve do so in spite of the surgical interference and not because of it.

Another result of adhesions is the obliteration of the foramina of Magendie. Through these foramina the ventricular or pial system of fluid communicates with the subarachnoid space. When the foramina are closed the fluid secreted by the pial covering of the choroid plexus is pent up in the ventricles. The bulk of this fluid then gradually increases. Its pressure becomes greater than the resistance of the brain tissue. The brain substance yields, the ventricular cavities distend, and hydrocephalus results.

Hydrocephalus is thus a possible consequence of every form of meningitis which does not quickly terminate in death.

The diagnosis of hydrocephalus is greatly facilitated by means of the x-rays. The treatment of the condition is surgical. Tapping of the ventricles at various points and attempts to reestablish the communication between the ventricles and the subarachnoid space are often made, and except in the case of puncture through the corpus callosum are usually futile.

Quinke and others have treated some of these cases by repeated lumbar puncture. One case is recorded in which by lumbar punctures, within two months, one and three-quarter liters of cerebrospinal fluid were withdrawn from a hydrocephalic three-year-old child. As much as 130 cm. were withdrawn at one sitting. After each tapping the pressure rose again in twenty-four hours.

OTHER VARIETIES OF MENINGITIS

A fully developed meningitis may ensue even in the course of diseases which primarily have their site of election outside the nervous system. A purulent meningitis may arise from the *Bacillus typhosus*, the pneumococcus, the influenza bacillus, or other organisms which usually invade the digestive or respiratory tracts.

Every energy should be devoted to elucidating the nature of the affection. The history, evidence of contact with cases of contagious diseases; examination of ears, throat, lungs, blood culture; examination of the blood for leukocytosis and for Widal's reaction; examination of the cerebrospinal fluid for leukocytosis, galactose and globulin content, and organisms—all may be necessary before a diagnosis is reached.

Any pus-forming organism can produce meningitis. The organisms which, apart from those that we have discussed, most frequently are implicated are the streptococcus and staphylococcus.

The streptococcal and staphylococcal meningitides are usually secondary to ear trouble or to trauma.

In children the mucous membrane of the middle ear is intimately connected with the meninges through the incompletely ossified petrosquamosal suture; hence, inflammation of the middle ear in children frequently implicates the meninges. To diagnose between otitis and meningitis is difficult. Because the former disease is often a cause of the latter it is always desirable, according to Lees and Barlow, "to have the tympanic membranes incised if the child be seen at or soon after the onset of the illness, and if the help of an aural surgeon can be obtained."

In purulent meningitis Kostlivy advises repeated cerebral puncture by the Neisser-Pollack method in order to determine where the pus is in greatest quantity. Over that spot the skull should be trephined.

The great object in all cases of meningitis is to begin treatment early. The initial treatment has already been outlined (see p. 293). Urotropin will tend to mitigate or to abort the disease. Lumbar puncture will relieve the symptoms and will give indications for more radical curative measures. Appropriate vaccines or antitoxins may favorably influence the progress of the disease. If at the first signs of meningeal irritation urotropin be used and lumbar puncture be practised, meningitis may be elevated among the most amenable of all nervous diseases to treatment.

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CHAPTER VIII

TREATMENT OF SYPHILITIC DISEASES OF THE NERVOUS SYSTEM

By SMITH ELY JELLIFFE, M.D.

INTRODUCTION

THE treatment of syphilis of the nervous system differs from the treatment of syphilis of other parts of the body in a few but important particulars. The external remedies which are of value in treating superficial syphilitic lesions find no application in syphilis of the nervous system. Furthermore, the treatment of certain syphilitic affections, tabes, and general paresis, obtains few of the brilliant results obtained even with the most chronic of syphilitic manifestations elsewhere.

Taken as a whole, the results of treatment of the nervous manifestations of syphilis are gratifying, notwithstanding the general fact that the nervous system when modified by syphilis suffers greater functional losses than other bodily structures. This is to be expected in view of the fact that neurones as cells are vastly more highly specialized than any other cells of the body and that interference with their conducting capacity is readily brought about.

In the treatment of syphilis of the nervous system, we are met at the outset with at least three problems. In the first place, one has to deal with a virus whose effects, mediate and remote, can be transmitted from parent to child. Hereditary syphilis of the nervous system constitutes a special complex entity and needs particular emphasis from the standpoint of treatment.

Acquired syphilis of the nervous system, early or late, makes up the bulk of the clinical manifestations which are met. It offers no significant variations beyond those noted.

Parasyphilis, metasyphilis, or whatever one may wish to call the manifestations, seen most strikingly in the broad clinical groups of tabes and general paresis, like hereditary syphilis, is also a problem of itself. It has its special rules, and particular difficulties, which at the present time are being attacked with much vigor and hopefulness.

Clinically, syphilis of the nervous system is so protean, that unless one is on one's guard, the significance of syphilis as an etiological factor may be entirely overlooked. The physician with his eye riveted upon a clinical picture, amyotrophic lateral sclerosis, a progressive muscular atrophy, or a protracted sciatica, let us say, and mindful of the somber prognosis given in the majority of his available guides,

might overlook the fact that in some of the patients syphilis is of etiological moment.

There is probably no field in medicine wherein similar diseased pictures may arise from as many differing causes as in the domain of the nervous system. Nor, on the other hand, where a single etiological factor may give rise to so many dissimilar clinical pictures. Hence, the complexity of the whole subject, and the need for iterating and reiterating the advice that in nearly all of the clinical pictures which have or have not been given descriptive terms in nervous or mental disease, the possibility of syphilis as a direct etiological factor or as a complication, or as causing confusion, should be borne in mind. Hence, the necessity for detailed and minute inquiries into all of the possible symptomatology, of hereditary or acquired syphilis, which in all questionable cases should be supplemented by complete serological and cytological examinations.

While one may with considerable confidence rely upon a completely negative history and negative or uncertain laboratory findings, yet in a small number of instances occult syphilis will escape both the anamnestic and laboratory nets and pass unrecognized. Increasing refinements in both directions are decreasing this number, however, and at the present time the possible syphilitic element rarely escapes detection if carefully sought for.

Turning our attention from the anamnestic search for the etiological factor as supplying the real point of departure for the therapeutic attack, it may be profitable to discuss in brief some of the historical landmarks in the development of our clinical, pathological, and etiological knowledge of syphilis of the nervous system.

It is only within comparatively recent times that the rich collection of apparently dissimilar diseases has been strung along on the one etiological string. The dissimilarities in clinical expression, even within the nervous system, have been so striking as to obscure the fundamental underlying factor.

The formerly very distinct dividing lines between the lesions of hereditary syphilis, acquired syphilis, and metasypilis are now, however, slowly being softened, and in time it may probably seem strange that it could have been thought that spinal and cerebral syphilis on the one hand should ever have been considered different, let us say, from tabes and general paresis on the other.

Since it has come to be believed that both tabes and general paresis rest upon a syphilitic basis, the variety of syphilitic disorders showing fairly clear clinical entities has been enlarged. Further, with the recognition of many acute and subacute psychoses due to syphilis the psychiatric borders have been further extended.

In the present chapter then the discussion will concern itself chiefly with clinical forms as much as possible, without any extended attempt to separate types, which in reality have so many intermediaries as to defy analysis.

At the same time it needs to be emphasized that such intermediary

forms are an ever-present reality. Classical pictures of a disease are largely literary chefs-d'œuvres. They are the product largely of the descriptive art. The actual processes going on in nature, the conflict of interest, *i. e.*, *treponema vs. homo*, shows not classical types but multiplicity of variations, with here and there statistical prominence of this or that trend in the reactive compromise.

This point of view, however, does not lend itself to didactic presentation, and precise directions are as necessary to the practising physician as are maps and time-tables to the traveller in a known or unknown land. It is, therefore, a concession to such necessities that in this chapter the syphilitic diseases of the nervous system which are to form the problem to be grappled with by therapy will be sketched in more definite and bald outlines than the real conditions permit.

Practical utility suggests then a consideration of those syphilitic disorders, first of the meninges, then of the cranial and peripheral nerves, the spinal cord, the medulla, midbrain, and of the brain proper.

Before plunging directly into the subject of the treatment of symptom groupings, which descriptive neurology, for practical purposes, has termed syphilitic meningitis, meningomyelitis, tabes, cerebral gumma, paresis, cerebrospinal syphilis, syphilitic endarteritis of the spinal or cerebral vessels and the like, it will prove of eminent practical value to trace in outline the older beliefs concerning the nature of syphilis and the modes of treatment. Then the modern period of pathological differentiation may be entered upon, the exact knowledge of the organism itself and its allies sketched, and the more precise efforts at destroying it, and of aiding the human body in its struggle considered in detail as the main object.

Historical Outline.—In just what places syphilis was an endemic disease before its great extension in the sixteenth century is a question that must be left for solution to the syphilographer (Sudhoff). Still it is not without great interest for the student of diseases of the nervous system to observe that syphilis of the nervous system received very little attention until the beginning of that same century. One can readily understand that this should be so if the hypothesis of the American origin be accepted. Such an acceptance seems almost inevitable in view of the lack of observation of syphilitic disease of the nervous system before 1500, and also the lack of observations relative to general paresis. This in spite of Sudhoff's recent finding of an Italian receipt book dated 1465, with receipts for the "French disease," which same form of receipt was used for the treatment of syphilis after its epidemic outbreak in Naples in 1494, and also the additional concomitant use of the name "French disease."

It is more or less immaterial, after all, just when and where this disorder arose. It is very certain, however, so far as Western medicine is concerned, that the first great epidemic extension of the disease began in 1493 to 1500, and that the study of its cause and its clinical variations began at that time.

As we are concerned solely with syphilis of the nervous system

we shall limit our historical remarks to those authors who have called attention to syphilitic involvement of nervous tissues. Naturally such a presentation cannot claim completeness nor could a detailed record serve any purpose other than that of bibliographical interest.

Bloch in his monumental monograph has brought together many interesting details, tending to show how well versed the South American peoples were in its manifestations, among which many concerning the nervous system are recorded. Indeed, if we are to accept the statements of Bloch, from evidence afforded by the Spanish physician Diaz de Isla, who was in practice in Barcelona when Columbus returned from Hayti, and who treated the first sailors, including the pilot, the Indians of Central America had a complete system of treatment for syphilis. The disease was named in several Indian dialects, among them the Aztecs, who we are told even in the early days distinguished the large pustular syphilides from that with smaller pustules. Mexican myths are also concerned with the disease. Seler the sun god was looked upon by the Mexicans as the main cause of venereal disease. Here, if we are to follow Kühn in his general interpretation of comparative mythology that after all the sun god, the giver of life, the transmitter of fire, the borer, or firemaker, and the male genitals were symbolically one and the same, it can be readily understood why such an idea was perfectly relevant, and possibly not far from the truth.

So far as the writings of ancient Greek or Latin writers are concerned, I am convinced that we find nothing to show that they saw any such pictures as began to be very prominent in the Sixteenth Century. Tornéry, who has discussed the descriptions of the early writers, does not enter into the question. Monkmöller, who has given the most thorough and comprehensive history of general paresis, concludes that the ancients never saw it, and that the descriptions of Aretaeus and Caelius Aurelianus, which have often been taken as descriptions of paresis, are probably the association of mental defect or of aphasia with hemiplegic attacks. Falk had come to much the same conclusion.

The era of definite nervous syphilis, however, began closely following the epidemic spread. Suffice it to say that within three or four years the disease had foci throughout the greater part of Europe.

Leoncino as early as 1497 described paralysis as a consequence of the disease. He here referred to what we would call hemiplegia, which may result, as is well known, within a few years, even a few months, after infection.

Joseph Grünbeck (1503), Emser (1511), Ulrich von Hutten (1519), all lay writers, mention paralyzes of the limbs as due to the disease. Emser speaks of his patient, a syphilitic paralytic, and with a psychosis, as having made a remarkable recovery under treatment by Bruno —by making a vow. Paracelsus (1530), although still confusing gonorrhea with syphilis, as had been and is still being done, left indications of a description of syphilitic meningitis, and in speaking of the syphilitic virus said that it affected all of the organs of the body. Nowhere in Fracastorius (1521), to whom we owe the name, are direct

references to the nervous system to be found. Nicolaus Massa (1556) gave an early description of syphilitic neuralgias. Borgarutius (1567) also described neuralgic pains due to syphilitic disease of the meninges. Amatus Lusitanus (1561) described headaches due to intracranial osteitis of syphilitic origin. Botalli (1563) made an observation that blindness might be due to syphilitic disease of the brain.

During the following century many references have been found showing the recognition of the relationship of syphilis to nervous disease. Only a few can be mentioned here. Thus, Guarinoni (1610) described epileptic attacks from syphilis of the brain. Vidus Vidius (1611) described epilepsies as due to syphilitic cranial caries. Thierry de Hery (1634) and Zechius (1650) also called attention to syphilitic spasms as well as epilepsies. Zacutus Lusitanus (1644) described cases of blindness due to gumma of the brain, quoting Botalli a century ahead of him. In 1696 a special treatise on syphilitic pains was written by Blagny.

Attention might be called to the works of Scholtzius (1610) and Willis (1672) apropos of the subject of general paresis. To Willis has always been ascribed the honor of the first description in which one could definitely recognize general paresis.

By the end of this (seventeenth) century an early broad view of syphilitic nervous disease had been obtained. Syphilitic headaches were described by Felix Plater (1641). Rhodius (1657) described gummata of the dura and syphilitic hemiplegias, Ballen (1663) spinal syphilitic disease and spasms in the facial region, Cummius (1684) diplopias and eye palsies. Astruc has reviewed these writings completely.

During the eighteenth century the picture expanded rapidly. Inter-costal neuralgias (1762), deafness, loss of smell, caries of base of skull (1762), sciatica (1745), psychoses. Syphilitic mania was described by Sanché in 1777, amaurosis (1748), facial palsy (1758), leptomeningitis (1766), syphilitic arterial disease (1766), paraplegias (myelitis) (1771), and a number of other conditions were described, and may be consulted in Lagneau's interesting monograph in which 234 case histories are collected. Astruc, Bonet, and Morgagni offer the richest literary sources.

It may be recalled in this place that John Hunter in 1787 stated that he never observed syphilis of the internal organs, including the brain. The weight of his authority retarded progress for many years, especially in England. Indeed, it was not until Ricord's sound observations were published that Hunter's enormous blunder was fully remedied.

Virchow's studies (1847) on phlebitis and arteritis laid the foundation for our modern knowledge of bloodvessel syphilis, although it may be recalled that Morgagni (1766) and Horne (1782) both made extremely pertinent studies on vascular syphilis. These have been fully developed by Huebner (1874) and Alzheimer (1904).

The studies of Virchow on the formation of gummatus granulomata and related syphilitic phenomena, practically established the modern era of study of the pathology of this disease.

The succeeding years have filled in the picture with a number of details, the chief additions having been those of Nissl and Alzheimer, who have established the highest criteria for the pathology of this disease so far as the nervous system is concerned.

The latest chapter in this interesting history is that dealing with the discovery of the exciting agent, and the final clearing up of the entire subject of etiology and modes of infection. Schaudinn (1905) demonstrated the parasite which he called *Spirochæta pallida*. Its varied synonymy to accord with principles of botanical and zoölogical nomenclature need not detain us. *Spironema pallida*, *Treponema pallidum* are those most frequently employed. Doele (1892) is thought to have first seen the parasite, but Schaudinn, then Epaschen, Fischer, Metchnikoff, and Roux established its identity and its affiliations.

It was soon found in congenital syphilis in the nervous system, in syphilitic gumma of the brain, and spinal cord, in syphilitic meningitis, even in the cerebrospinal fluid, both of congenital and acquired syphilitics. Moore (1913), Noguchi, Nichols, and Hough have found it in the paretic brain, and it probably will soon be found in the spinal meninges of tabetics.

The final studies of Neisser, Metchnikoff, Roux, and others have laid bare the entire story of the inoculability of the disease and its transmission from animal to animal, while, utilizing the knowledge gained by Bordet and Gengou, Wassermann and his pupils have elaborated a serobiological technique which has made one independent of clinical or anamnestic data relative to a knowledge on the part of the patient of the infection.

Thus, in hardly more than seven years, a flood of light has been thrown upon the disease and its relation to other infusorial-caused diseases, notably trypanosomiasis, which has illuminated and made clear the entire path which has been so busily travelled since Columbus brought back this most portentous exotic to the old world.

Diagnosis.—The diagnosis of syphilis of the nervous system presents certain difficulties which rapidly increasing perfections in laboratory technique are resolving with considerable success. These laboratory clinical findings combined with those of the neurological status permit an almost certain diagnosis of this disease in the nervous system, either as congenital, acquired, or as para- or metasyphilis.

The chief features in such a scheme of diagnosis are: (1) Search for the organisms; (2) serological investigation of the blood and cerebrospinal fluid; (3) cytological examination of the cerebrospinal fluid; (4) chemical examination of the cerebrospinal fluid; (5) clinical examination of the pupillary reflexes.

1. **Search for Organisms.**—The parasite has been found in the cerebrospinal fluid, but as yet in but few instances. It has been cultivated from the cerebrospinal fluid (Nichols and Hough). Increasing experience along these paths of study will probably develop important diagnostic aids. These are, as yet, of less practical use than others to be enumerated.

2. Biological or Serological Tests of Blood and Cerebrospinal Fluid.—

(a) **BLOOD.**—This is not the place to discuss the technique of the Wassermann reaction. Yet an adequate technique is extremely important. The consensus of opinion lays stress upon the superior reliability of the original forms of the technique. Practically all forms of early syphilis of the nervous system should show a positive Wassermann reaction in the blood. There are exceptions, but syphilis of the nervous system, like syphilis elsewhere, gives a positive reaction in early cases. Scarlet fever, trypanosomiasis, frambesia, a few cases of multiple sclerosis, these may occasionally give a positive blood Wassermann.

Whereas, the percentage of positive findings should be as high as 100 per cent. in cases of early syphilis of the nervous system, in its secondary and tertiary stages the percentage of positive results may fall to even 70 per cent., and in the latent period may sink to 50 per cent. Whether these reduced percentages speak for the good results of treatment or are dependent upon other as yet uncertain factors is yet to be decided. The alcohols interfere with the reaction.

Citron and a number of other workers in this field have repeatedly stated that the cure of syphilis, nervous as well as otherwise, bears a direct relationship to the results obtained by the biological tests. This is an important feature in determining the therapy, and merits close attention. It has been frequently found, however, that after a period during which the blood results have been negative there has been a sudden return of the reaction. From this many students argue that the result of a Wassermann test of the blood is of little value in determining the control of antisyphilitic therapy.

On the other hand, some of the most competent students of the biological reactions are disposed to be largely guided in their therapy by the results of the Wassermann test. Brück has summed this up somewhat as follows: "Study of the Wassermann reaction shows that energetic antisyphilitic treatment should begin as soon after infection as possible, until the blood reactions are negative. One should then carry on a regular series of blood controls, and as soon as a return of the reactions shows itself, energetic syphilitic treatment should be inaugurated. One is justified in not pushing antisyphilitic treatment only when the blood reactions have been persistently negative for a considerable period of time."

This whole subject of the relationship of the reaction to treatment is well discussed by Plaut, where all of the pros and cons may be found. We have also discussed it in the section on therapy.

(b) **CEREBROSPINAL FLUID.**—The behavior of the cerebrospinal fluid to the Wassermann reaction is of special significance in the diagnosis and treatment of syphilis of the nervous system. It is almost uniformly positive in general paresis, even when small quantities (0.2 c.c.) of the serum are employed. By the use of such small quantities it would appear, from the work of Hauptmann and Hössli, that paresis alone will cause a positive result, but with larger quan-

tities of cerebrospinal fluid (0.4 to 0.8 c.c.), then practically all forms of cerebrospinal syphilis will give a positive reaction; tabes, cerebral syphilis, meningomyelitis, etc. (Hauptmann, Holzmann, Swift, and Ellis.)

Syphilis without nervous symptoms uniformly gives a negative reaction, even when large quantities of the liquor are used.

It might be mentioned here again that multiple sclerosis occasionally gives a positive Wassermann reaction in the blood, and this apparently independent of any syphilitic history, and with other syphilitic findings negative. Kaplan's and Casamajor's findings are negative for a series of neurological cases, amyotrophic lateral sclerosis (Spiller has reported cases of this syndrome with syphilis probable), chronic anterior poliomyelitis among them.

We cannot go into the full analysis of the findings of the Wassermann reactions in the cerebrospinal fluid in this place. The chief features are here didactically outlined. It must be constantly borne in mind in the diagnosis of syphilis of the nervous system that the reaction of the cerebrospinal fluid in the Wassermann test is purely monosymptomatic. The positive or negative results must always be interpreted by association with other laboratory and clinical tests. As Nonne has well said, the Wassermann reaction is only a symptom. Like other symptoms in a syndrome it may or may not be present without affecting the validity of the syndrome from its diagnostic aspects.

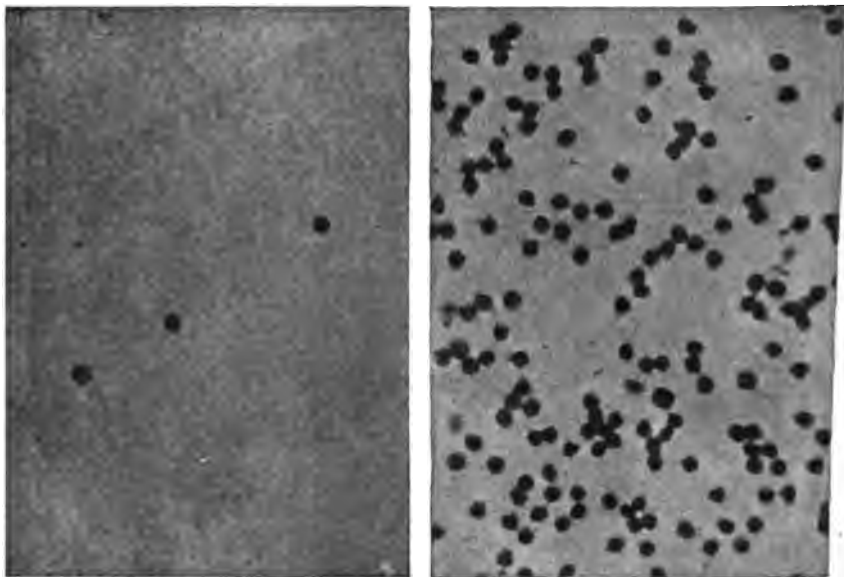
3. Cytological Examination of the Cerebrospinal Fluid.—Quinke, by his procedure of lumbar puncture, opened a new field for laboratory diagnosis. Applied to the study of the cellular as well as the chemical constituents of the cerebrospinal fluid it has rendered the diagnosis and therapy of syphilis of the nervous system an immense service. The technique of puncture cannot be entered into here, but one point should be borne in mind: the procedure is not always without danger. It should be done with care, the fluid being withdrawn very slowly, drop-wise in some cases, and the patient should rest in bed several hours, preferably both before and after the operation. One of the functions of the cerebrospinal fluid is to maintain an equality in the intracerebral pressure, and any sudden alteration, such as is produced by the withdrawal of 5, 10, or 15 c.c. of fluid, is apt to disturb such equilibrium. Headache, nausea, vomiting, dizziness, are among the unpleasant effects in those who react badly. Such are few, but they exist, and care is imperative. Some are helped by lying quiet, with foot of bed elevated and with aspirin, 10 grains, repeated if necessary. In patients in whom brain tumor is suspected special care should be taken, as here a few cases of sudden death have been reported following lumbar puncture with withdrawal of fluid.

It is one of the drawbacks in cytological work with the cerebrospinal fluid that degenerative changes in the cellular contents take place within comparatively short periods of time. This shows itself particularly in the poor staining qualities of many of the cells and also in deformities and destructions. Whether this is an autolysis or what not

is as yet uncertain. At all events the cytological examination should be conducted as soon as possible after the withdrawal of the fluid.

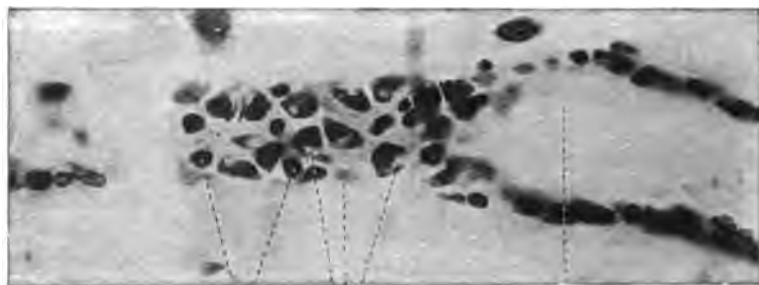
In pathological cerebrospinal fluid an increase in lymphocytes is the most striking feature. Over 7 to 10 lymphocytes to the c.mm. indicates pathological fluid. Other cells may also be found, such as

FIG. 17



Lymphocytes in normal (left) and paretic (right) cerebrospinal fluid. (Kraepelin)

FIG. 18



Lc lymphocytes; *Pc*, plasma cells; *L*, lumen of vessel.

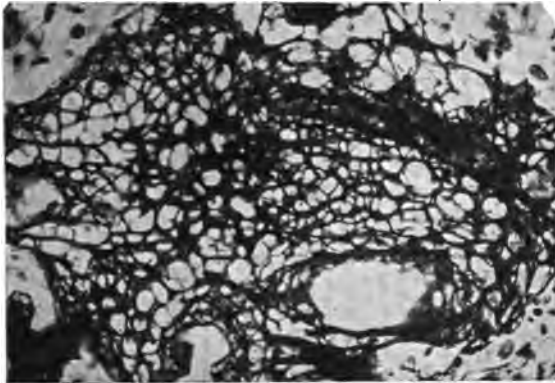
polymorphonuclear leukocytes, plasma cells, and occasionally eosinophile and endothelial cells. Red blood cells usually come from the wound of the puncture. We are not now considering the possibilities of traumas.

As to the origin of the pathological cells, authorities are not in accord.

Merzbacher holds that they are derived from the walls of the pial vessels, and from those of the cortex. The role of the ependymal vessels is also to be considered. Thus in parietic ependymitis a fairly constant picture in paresis, the cellular content of the cerebrospinal fluid is usually very rich in all of the adventitial cells mentioned.

In both tabes and paresis the infiltration of the meningeal tissues of brain and spinal cord by both plasma cells and lymphocytes is well known. Fischer, Nissl, Alzheimer have shown these (Nissl, Alzheimer loc. cit.). E. Meyer has demonstrated closely related findings in cerebrospinal syphilis. In the tertiary stages these infiltrations seem to occupy the more superficial structures of both the cord and cortex, whereas in both tabes and paresis the infiltration by cellular elements is deeper and more general, occupying for the most part the perivascular spaces or their immediate surroundings (Alzheimer, loc. cit.).

FIG. 19



Organisation of Virchow-Robin lymph spaces and penetration of ectodermal tissue by outgrowth of adventitial fibers. General paresis. Tannin silver. (Achucarro.)

The fluid is best fixed and imbedded, and then stained by the Alzheimer method. This is the most complete and satisfactory method thus far devised, as it permits of a counting and study of all of the cells. The Fuchs-Rosenthal counting chamber, however, is that used in the greater number of instances. Its results are more quickly obtained, although they lack the finality of the Alzheimer method.

A normal cerebrospinal fluid is a clear liquid. It has a specific gravity of 1006, a slightly alkaline reaction, and is about free from cellular constituents—1 to 5 lymphocytes per c.mm. as estimated by the Fuchs-Rosenthal method may be considered normal. (See Thomsen, Hill, Halliburton, *Proc. Roy. Soc.*, vol. lxiv, for normal fluid.)

Changes in Cerebrospinal Fluid.—In cerebrospinal syphilis, in tabes, and in general paresis there is usually an increase in the number of lymphocytes. They may run up into the hundreds, especially in some cases of acute meningomyelitis; also in some fulminating cases of paresis.

In fact, most meningitic processes are accompanied by increase in cellular content. Polynuclear cells are frequent in the acute types, sometimes tuberculous meningitis excepted, and as the process tends to chronicity there is a tendency to lymphocytosis and loss of other cell types. The pleocytosis of syphilis has usually been attributed to a meningitic process. It has been suggested as due to a periarteritis as well (Szecsi).

Certain general variations may be recorded. The earlier students were more didactic in their statements concerning specific differences in the number of cells as distinguishing cerebrospinal syphilis, tabes, and paresis. Further extension of the studies shows them to have been in part unwarranted. Thus it has been said that the lower numbers point to cerebrospinal syphilis, the higher number to tabes, and the highest to paresis. This is perhaps so, but it is not an absolute rule. The number of cells seems to bear a more definite relation to the activity of the underlying irritation or inflammatory process than to its kind. Thus a stationary tabes may show few cells, also a paresis in remission, whereas an acute cerebrospinal syphilis or an acute meningomyelitis may show many cells.

A fluid rich in polymorphous cells is indicative of a very active process, syphilitic or otherwise.

In cerebrospinal syphilis, tabes, and particularly in paresis, it is of great importance to note that a pleocytosis, oftentimes of very marked grade, may antedate all neurological symptoms of the after-coming disorder. This has been shown repeatedly by Sicard, by Ravaut and others. This preparetic period has been diagnosticated as long as two years before the onset of the disease. This point is well to bear in mind when one's advice is asked, as to the advisability of marriage of syphilitics in the forties. It is not at all an infrequent experience to find the outbreak of paresis occurring in men of from forty to forty-five, who have been from one to three years married, and many have waited thus long, feeling that because of an early syphilis it were wise to defer marriage until a safe period. A return will be made to this prophylactic feature later in the more detailed discussion of the therapy.

How long after infection by syphilis may lymphocytosis appear in the cerebrospinal fluid? Varying answers are available, but Ravaut has reported their presence at least two months after infection. Their presence almost invariably speaks for nervous syphilis. In patients who have thus far been followed, the development of nervous accidents, hemiplegia, paraplegia, meningomyelitis, etc., has taken place.

With active therapy the pleocytosis is apt to disappear. Those patients who retain their lymphocytosis, are they on the way to tabes, or paresis? This is an important problem which has been answered Yes and No. Its definite answer is yet to come, as the necessary time during which the full significance of the results of cytological study of the fluid has not yet elapsed.

From the therapeutic point of view then the whole subject of pleo-

cytosis of the cerebrospinal fluid is full of significance, and in need of constant attention. In this connection it should again be emphasized that lymphocytosis alone does not mean syphilis alone. It can be said that absence of lymphocytes at least negatives tabes and paresis—to this generalization there are but few recorded negative observations. (Klieneberger, *Archiv f. Psychiatrie*, 1911; Foster, Lewandowsky, *Handbuch der Neurologie*.)

Lymphocytosis is not limited to syphilis of the nervous system, however. It is marked in sleeping sickness, an allied disease (Spielmeyer, Mott). It is often also high in tuberculous meningitis, but is here usually complicated by the presence of polymorphonuclear leukocytes. In a few cases of multiple sclerosis lymphocytes have been found. In the acute stages of poliomyelitis lymphocytes may be found, also in cerebrospinal meningitis and in herpes zoster.

As has been previously noted for the biological tests, so the cytological tests are only monosymptomatic; alone they are not absolute, but taken in combination they are invaluable in both diagnosis and in treatment.

In treatment particularly it would seem then that the consensus of opinion points to the fact that the grade of lymphocytosis is a fairly accurate guide to the severity of the process. In cerebrospinal syphilis particularly a steady diminution in the number of lymphocytes goes on *pari passu* with the efficacy of the treatment. This is not an absolute rule in the metasyphilitic affections, for here a diminution in lymphocytes very frequently may take place without, however, any corresponding alleviation of the tabes or paresis. In certain cases of tabes with pain, diminution of the pain with slight reduction in the number of lymphocytes has been observed following antisiphilitic therapy.

This diminution in the number of lymphocytes has taken place with mercurial as well as arsenical (salvarsan) therapy.

Only the more striking features of our knowledge regarding the cytology of nervous syphilis can be here presented. The literature is extensive, and contains many aberrant findings. A wide reading counsels one to take every fact into consideration, clinical as well as laboratory, and to avoid laying too much stress upon any single fact or closely related group of facts.

4. Chemical Examination.—Here the presence of a reducing agent (Fehling's) and of globulin is to be estimated. Most liquors contain the former. When present in large quantities it may point to a tuberculous meningitic process.

Increased globulin content is a characteristic feature of paresis. In tabes increased globulin is the rule as is also the case in cerebrospinal syphilis, but to a less extent. Markedly increased globulin content is not infrequent in spinal cord tumors, gummatous or non-syphilitic. The globulin reaction is apt to run along with the lymphocytosis. It has no apparent alliances with the findings of the Wassermann technique.

The whole question of increased globulin content is in need of more

thorough revision, but in general it may be said that the matter may be summarized as has already been done. It might be added that either a mild or strongly positive globulin increase may be observed in tuberculous meningitis and other meningitides, pneumonia, influenza, etc. A marked increase in globulin with negative Wassermann and negative pleocytosis speaks not only for a tumor, as has been noted, but also for any cause of compression of the spinal cord.

Summary of Laboratory Findings.—**FOUR REACTIONS.**—Before passing to the diagnostic significance of the clinical examination of the eye reflexes, a word should be said relative to the value of these “four reactions,” as Nonne has called them. It has been said that taken alone they may mean nothing positive, so far as a differential of the different types of syphilis of the nervous system is concerned, but when read together they afford important guides to diagnosis and to treatment.

Nonne's conclusions are perhaps the most extensive that we possess on this point. They are founded upon his own experience with 167 patients with tabes, 179 with paresis, 97 with cerebrospinal syphilis, 68 patients with multiple sclerosis, 38 with brain tumor, and 14 with spinal cord tumor.

Expressed schematically, which schemes, as he well remarks, are not products of nature but of man, the following results of the four reactions are to be expected:

(1) Blood and (2) Fluid.

I. Blood examination.

Wassermann reaction.

- (a) Positive. Is characteristic of syphilis with few exceptions (already noted).

A positive Wassermann of the blood serum says nothing further than that the individual has come in some manner in contact with syphilis, either through heredity or by infection. It does not say that the disease from which he suffers is due to the syphilis.

- (b) Negative. Is differentially diagnostic against paresis, since it is only very rarely that the blood in paresis gives a negative reaction.

II. Study of Cerebrospinal Fluid.

- (a) Normal Fluid. Pressure 90 to 130 mm. Water globulin reaction negative—not over 5 or 6 cells to c.mm. (Fuchs-Rosenthal).

(b) Pathological fluids.

1. Increased pressure—over 15 c.mm. water.
2. Positive phase I. Globulin reaction.
3. Increased cell count.

These three symptoms, in coördination or alone indicate the presence of an organic nervous disorder, syphilitic or non-syphilitic.

- (c) If the disease of the nervous system is syphilitic, then the Wassermann test of the fluid will show. If the Wassermann reaction, original method (0.2 c.c. of the fluid) is positive, there is great probability that the patient is a paretic, or a taboparetic, much less often a cerebrospinal syphilitic, or a pure tabetic.

In nearly all cases of cerebrospinal syphilis and of tabes the Wassermann reaction becomes positive by using 0.4 to 1 c.c. of fluid.

Nonne's typical findings are as follows:

I. Paresis or taboparesis.

1. Wassermann reaction in blood positive (100 per cent.). Pressure increased.
2. Phase I, globulin reaction positive (95 to 100 per cent.).
3. Lymphocytosis (95 per cent.).
4. Wassermann in fluid.
 - (a) Positive—about 85 to 90 per cent. with original method and 0.2 c.c. fluid.
 - (b) Positive in 100 per cent. with larger quantities of fluid.

II. Tabes without paresis.

1. Wassermann reaction in blood—positive 60 to 70 per cent., pressure usually increased.
2. Phase I, reaction globulin and positive (90 to 95 per cent.).
3. Lymphocytosis positive (90 per cent.).
4. Wassermann in fluid.
 - (a) Original method, 0.2 c.c. positive 5 to 10 per cent.
 - (b) Larger quantities, 100 per cent.

III. Cerebrospinal syphilis.

1. Wassermann reaction in blood positive (80 to 90 per cent.). Pressure frequently increased.
2. Phase I, reaction usually positive, exceptionally negative.
3. Lymphocytosis nearly always positive.
4. Wassermann in fluid.
 - (a) Original methods (0.2 c.c.) positive in about 10 per cent.
 - (b) Larger quantities nearly always positive (of value in diagnosis of multiple sclerosis, cerebral and spinal tumor).

These results of Nonne's summarize fairly accurately the present-day attitude on the value of the four reactions. The full significance of the findings can be gained only by reference to the originals. This field of work is rapidly expanding, and that which now appears on the frontiers of our knowledge will undoubtedly be much modified by the rapidly advancing army of investigators.

Among other things it may be gathered that most of the therapeutic studies which have appeared, and which bear little or no evidence of the utilization of even the laboratory methods here sketched in outline, are of very little value. This is particularly true in the field of tabes, paresis, and cerebrospinal syphilis. A number of papers have appeared

from the pens of able students of neurology and psychiatry which are comparatively worthless because of the uncertainties of diagnosis, lacking the final word of laboratory technique. It is not the place to point these out specifically—here only the more important guiding principles of critique which should be utilized in the valuation of such studies can be laid down.

5. The Eye Reflexes.—In the diagnosis of syphilis of the nervous system the neurological examination of the eye reflexes is of paramount value. Here irregularity in the size of the pupils, irregularity in the pupillary margins, the impairment of the consensual light reflex, the slowing in reaction to light, fatiguability of the light reflex, alterations in response to accommodation efforts, the full development of the Argyll-Robertson syndrome are all to be considered. These, one or all, constitute extremely delicate and valuable criteria for the clinical appraisal of syphilis of the central nervous system.

A fully developed Argyll-Robertson syndrome—loss of direct pupillary light reflex, with free and ample response to accommodation reflexes in one or both eyes—represents for the most part a fairly positive criterion of syphilis of the nervous system.

There are many who believe that this syndrome affords absolute proof of nervous syphilis. This we do not believe to be true, not only upon clinical, but also upon anatomical grounds. Clinically the Argyll-Robertson syndrome has been observed following direct injury of the midbrain structures (pistol shot, Guillain). It has been observed in poisonings other than those of syphilis, alcohol, in Korsakow, Wernicke's polioencephalitis superior. It may result from pressures, tumors of third ventricle, pineal, from poliomyelitis, from trypanosomiasis, and from other rare and anomalous disorders. Anatomically the syndrome represents implication of certain reflex paths in certain peculiar combinations, and such implications and combinations are purely fortuitous and accidental, *i. e.*, so far as nosology is concerned.

As a matter of fact, however, these combinations rarely take place except as a result of the widespread changes induced by one particular type of poisoning—the syphilitic virus—so that for clinical purposes the presence of a permanent, bilateral, Argyll-Robertson syndrome is nearly enough positive for syphilis to permit one to assume its presence, and to therapeutically guide one's self accordingly (Rose).

Here again, however, one meets with the pertinent suggestion that the testing for the Argyll-Robertson syndrome is not as simple as it is usually supposed. The ordinary devices of having a patient face the window, and then cover and uncover the opened eyes with the hand; focussing the eye upon a distant object, and then upon the finger in close proximity to the nose; these tests for the most part are entirely too crude to permit one to judge with certainty that the Argyll-Robertson syndrome is present. Such methods may suffice for the majority of instances, but in no field of neurology is it more desirable to utilize the most accurate methods than in dealing with the vexed question of syphilis of the nervous system.

Few clinicians can follow out the intricacies of Weiler's complicated methods, but they may be necessary in certain doubtful cases.

Repeated examinations, under carefully regulated supervision, in the daylight, and in the dark-room, are therefore desirable when testing for anomalies in the pupillary reflexes. Careful checking of the results obtained by the small pocket electric lamps is imperative, as occasionally they give anomalous results and may lead to serious errors (Oppenheim). The presence of a fully developed Argyll-Robertson syndrome may be said to be preponderating proof positive of syphilis of the central nervous system, particularly of the cerebral and mid-brain neurones. Its absence, however, by no means negatives syphilis of the nervous system, since cerebral gummata, cerebral syphilis, paresis, tabes, syphilitic meningitis, meningomyelitis, cord gummata, syphilitic radiculitis, syphilitic neuritis, all may be present without any anomalies in the pupillary reflexes. It has already been pointed out that the laboratory findings of paresis and of tabes have antedated the development of neurological signs by at least a couple of years, and, moreover, it has been emphasized that pathological alterations in these pupillary reflexes are after all only chance happenings, that certain reflex arcs in certain combinations are caught in the mesh of the infiltrative, syphilitic alterations. The chance is a large one, it is true, but still it is purely a statistical matter of what has happened.

As this chapter deals with therapy chiefly, and diagnosis only in so far as it is related to therapy, a fuller consideration of the pupillary reflexes is unnecessary. One word may be added: in many cases of cerebrospinal syphilis one can gauge the progressive amelioration of the patient's condition by the gradual return of the anomalous pupillary reflexes to a more normal condition. Thus an absolute Argyll-Robertson syndrome may become a relative one. A unilateral Argyll-Robertson may be lost; slowly reacting pupils may show prompt reactions, irregularities in size may disappear; rapid fatiguability may discontinue; a consensual light loss, often the first anomaly to appear in cerebrospinal syphilis, will clear up; irregularities in the pupillary outlines will make way to regular outlines, etc.

An inability to modify pathological pupillary reactions by ample syphilitic therapy argues in part for the chronicity of the process, and the inefficacy of the treatment. This is not an absolute rule, however. It may be possible for a syphilitic process to permanently destroy portions of the pupillary reflex paths, and then be completely and permanently arrested. The pupillary reflexes, however, remain impaired.

In relation to this question of the pupillary reflexes, and antisymphilitic treatment, the problem arises—what is the probable outcome of a syphilitic process which comparatively early in its course has destroyed the pupillary reflex paths? Can it be decided say after two, five, or ten years, during which time there has existed an Argyll-Robertson syndrome and little else, that the disease has been completely and permanently arrested? Since the rapid extension of knowledge

concerning the cerebrospinal fluid this question can be answered better at the present time than ever before.

If the signs of a meningitis—acute, subacute, or chronic, *i. e.*, increase in cellular elements, increase in globulin, and positive cerebrospinal fluid—Wassermann remain absent, then it can be taken as highly probable that the disease process has been arrested. Judging by clinical methods alone, Nonne is of the opinion that a long-standing and unchangeable Argyll-Robertson pupil may be the only lesion in a practically cured syphilis of the nervous system. It is, however, to be realized that such a pupillary anomaly may exist as long as from twelve to sixteen years—alone—and then the patient will develop a paresis or tabes. Of 11 personal observations now existing from ten to twenty years, only two syphilitic patients with long-standing Argyll-Robertson pupil have not developed further signs of brain syphilis. Certain deep-seated, chronic, syphilitic arterial processes, which may lead to focalized lesions, hemiplegia, aphasia, etc., may, however, go on for some time without distinct signs of meningeal irritation with the characteristic cellular reactions.

The whole situation of latent syphilis is by no means cleared up, even with the flood of light thrown upon it by the biological and cytological studies just referred to, but that it is approaching solution there is every reason to confidently expect. A return will be made to this question later in discussing the active therapy.

Clinical Forms.—It has already been stated that the clinical forms of syphilis of the nervous system are largely abstractions. The pathological processes are predominantly either meningeal, arterial, or infiltrative, *i. e.*, gummatous in character, alone or in combinations, and the clinical manifestations are extremely variable, complex, and confusing, depending upon the interactions of the pathological trends and the variations in anatomical paths interfered with.

Fortunately, for a general outline of therapy, the clinical type is perhaps of secondary consideration, yet there are certain therapeutic variables that render it desirable that a fuller analysis of clinical forms should be made than would at first sight seem advantageous. For instance, this viewpoint gains prominence when it is recalled that certain patients with meningitic infiltrations of the base, with or without gummatous nodules, either of the base or of the convexity, may be at times clinically indistinguishable from paresis. A nihilistic therapeutic attitude relative to the latter process would therefore work greatly to the disadvantage of a patient with the former. A like situation, though perhaps less frequently, is present in primary syphilitic endarteritis of the vessels. That type of syphilitic endarteritis of the small vessels of the cortex to which Alzheimer has called particular attention is another case in point.

It therefore seems desirable to pass rather rapidly in review some of the more salient "types" or "clinical forms" of syphilis of the nervous system, again calling attention to the more or less artificial character of "type" or "form" creations.

In the discussion then we shall take up the following *clinical forms*:

1. Syphilis of Cranial Bones, p. 321.
2. Syphilis of the Basal Meninges, p. 322.
3. Syphilis of the Convexity; Epilepsies, p. 323.
4. Cerebral Syphilis—Arterial Types, p. 324.
5. General Paresis, Taboparesis, p. 342.
6. Tabes, p. 369.
7. Syphilitic Psychoses, acute and subacute, p. 406.
8. Syphilitic Spinal Meningitis; Meningomyelitis; Myelitis; Syphilitic Radiculo-neuritis, and related Syphilitic Syndromes, p. 408.
9. Hereditary Syphilis of Nervous System, exclusive of Paresis or Tabes, p. 415.

For a fuller consideration of the protean variations the monographs of Rumpf, Nonne, Mott, Plaut, Oppenheim, Forster, Schaffer, and Fournier should be consulted.

1. SYPHILIS OF THE CRANIAL BONES CAUSING NERVOUS SYMPTOMS

These were recognized as early as the end of the sixteenth century, as has been noted in the brief historical sketch here presented. It may be recalled also that one of the evidences speaking for the American origin of syphilis is the presence of a bony syphilis in some of the prehistoric cranial bones found in various Central, South American, and North American localities (Hansemann, Orton, Herxheimer, Bloch).

Syphilis of the cranial bones shows itself practically in the form of gummata. Caries of the cranial bones alone, while known, rarely gives rise to nervous symptoms, headache excepted. These gummata may be circumscribed, in which case, if large enough, they give rise to the symptoms of a tumor of the brain, which signs are largely determined by the precise location of the gummata. These circumscribed gummata may attain enormous proportions. A personal observation recalls a gumma of the left frontal region (the size of a tennis ball), which originated in the bone dura and protruded into the right frontal lobe. Similar gummata are not infrequent.

Cranial bone gummata, usually involving the dura as well, are more frequently flattened and spreading. Here the symptoms of brain tumor are usually present. Headache, nausea, vomiting, sleeplessness, are among the general symptoms, while localizing signs in great variety, depending upon the situation of the gummatous masses are present. Epileptiform convulsions, lasting for years, may be the sole signs of such gummatous formations, occupying or due to pressure upon the motor areas. Such patients are often mistakenly treated as epileptics, and the monographs of Mott, Oppenheim, Rumpf, Nonne, in recent years, are replete with autopsy records of such cases. Monoplegias of various sorts result from such, as also aphasias, word blindness, and various cranial nerve palsies.

Rarer cases of cranial bone caries of the base—sphenoid—complicated often by caries of the upper vertebræ, are also known. Petren has studied these in detail, and has shown the value of the x-ray in their diagnosis.

2. SYPHILITIC MENINGITIS OF THE BASE

This is the most common form of cerebral syphilis. Its most frequent site, in the beginning, is in and about the interpeduncular space, thus almost invariably involving the optic chiasm. From here it tends to spread in all directions, pressing into the sulci, thickening the meninges, by infiltration, by arterial disease, or by gummatous growth. Usually all types of pathological alteration are found. The gummatous masses not infrequently invade the brain structures as well, grow about the emerging or entering cranial nerves, and even involve the bones of the skull, and the upper cervical vertebræ. Thus, spinal meningeal infiltrations almost invariably accompany this basal syphilitic meningitis.

Whereas the diffuse, conglomerate types are more frequent, isolated vascular disease, circumscribed gummata, or other simpler manifestations of the disease may occur, in which latter case the syndromes are apt to be simple.

The clinical course of the more frequent types of basal syphilitic meningitis often resembles general paresis, especially in the beginning, but the gradual extension of the infiltrating or gummatous developments introduce variants which often permit a differential diagnosis.

Headache is a frequent and early sign, often preceding other symptoms by weeks, months, or even years. It has the not infrequent night exacerbations so frequently pictured as characteristic of syphilitic headache. It is described variously as boring, stabbing, and percussion at the base, may show tenderness, though less frequently than in convexity meningitis. The cervical complications spoken of often result in stiffness of the neck.

An early implication of the optic nerve is to be expected. It shows itself (20 per cent. to 40 per cent. of the cases) either as a pressure neuritis (neuritic atrophy) of the nerve in one disk, then in the other, later, if headache and vomiting or other signs of intracranial pressure are prominent, choked disk in both eyes is apt to be present. Atrophic degeneration, optic neuritis, is less often found. Marked diminution in visual acuity may be present without any disk evidences of disease.

The third nerve is frequently and usually irregularly involved. It is characteristic of basal syphilitic meningitis that successive branches are implicated. First one eye may show a ptosis, then perhaps an internal rectus palsy, then the other eye may show a dilated pupil, slow in its reactions to light, then ptosis develops here. Occasionally the accommodation reflex is lost. A series of cases will show a great variety of oculomotor palsies. A true Argyll-Robertson syndrome is not infrequently obtained. (See Plate XIX.)

PLATE XIX

Fig. 1



Cerebral Syphilis Ptosis. Third Nerve Palsy.

Fig. 2



Pseudobulbar Palsy from Syphilitic Disease.

Fig. 3



Cerebral Syphilis. External Rectus Palsy.

Fig. 4



Abducens Palsy in Cerebrospinal Syphilis.

Other cranial nerves are often included. Variations in the corneal reflex, in the sensibility, pain, anesthetics of the face, point to a trigeminal complication. A peripheral facial palsy may be present. In some individuals the deeper lying cranial nerves 9, 10, 11, 12 are caught in the syphilitic extension, with their characteristic symptoms.

The mental picture is usually very striking. It is frequently that of a slowly developing apathy, or heaviness advancing to coma, or unconsciousness, with periods of acute confusion, possibly violent delirium. There is a marked variability in these patients from day to day and also considerable differences in different patients. Some patients develop a sort of drunken delirium; others are heavy, and stupid and apathetic; others are furiously violent.

One special feature is frequently met with. This is a rapid alteration in the mental picture, when after a period of acute confusion or deep coma the patient becomes almost practically clear within a few hours. This occurrence may even follow a period of convulsive seizures. Careful tests reveal an underlying series of defects, it is true, but from a lay viewpoint the patient may appear to have made a complete recovery so far as his psychosis is concerned. Without treatment, however, the patient again develops his apathy, confusion, delirium, or coma and not infrequently dies in this state. Sometimes death results by suicide as the patient develops, slowly or rapidly, a distinct depression with possibly persecutory ideas.

Thus, mentally, the patients may show the old-time rubrics of acute confusion, dementia, mania, melancholia, paranoia, etc. This alone indicates the futility of regarding the symptom pictures which have gone by these names as diseases, some for hundreds of years. Happily present-day psychiatry, largely under the influence of Kraepelin's teaching, recognizes them as only the protean and kaleidoscopic picture formation of not only syphilis, but other disease processes as well.

Biological and cytological methods have permitted this definite change in attitude, and have shown the essential and close relationship of many diverse neurological and psychotic syndromes.

3. SYPHILITIC MENINGITIS OF THE CONVEXITY

This differs from the former only in the trend of its symptoms. The pathological processes are practically identical. Many individuals show that the process is general, involving both the base and the convexity; in some instances the pathological changes being more marked on the convexity than on the base. It is largely a question of chance, the causes for which are as yet difficult to grasp, why one region should be affected more than another. Sometimes a blow on the head determines that general place as the site for the after-coming storm. This does not mean that the blow had much to do with the storm, it only serves as a cause to determine just where it will be more likely to centre.

Convexity syphilis, like the basal variety, may be a fairly localized

affair, or it may be diffuse. It may be limited to the meninges, or involve the bones, or the brain, or, as is most usual, all three.

Here, headache is a prominent sign. It is paroxysmal, and often shows a nocturnal increase in severity. Percussion affords valuable evidence, as localized tenderness is very frequent. Here the general symptoms of brain pressure are usually less emphasized. Nausea, vomiting, giddiness, may be present, but are usually late in development, or more transitory. Optic nerve changes are less frequent. Isolated symptoms are more prominent. Epileptiform convulsions indicate that the process is in or about the motor area. Not infrequently the attacks are of the Jacksonian type. Involvement of Broca's convolutions produces temporary or more enduring motor aphasic attacks. Sometimes these aphasic attacks clear up in a few minutes, an hour or so, or a few days. Minor speech difficulties may only indicate the possibility that an aphasia would develop. Complete motor aphasia develops only as a rule with hemiplegic or monoplegic accompaniments. Pseudobulbar palsy attacks indicate a bilateral involvement, probably both cortical, less frequently cortical on one side, and subcortical on the opposite side.

Monoplegias of varying types are not infrequent. Bergmark (*Brain*, 1911) has devoted a large monograph to this study. Sensory disturbances, hemianesthesiæ, astereognosis, haptic hallucinations are met with.

With diffuse meningo-encephalitic changes the picture of general paresis is assumed, and it is particularly difficult to differentiate this disorder. The clinical pictures may be as various as those of paresis. Possibly the only means that we possess to distinguish is that claimed by Plaut, and apparently substantiated by Nonne and several others, that in paresis the four reactions are all positive; the cerebrospinal fluid showing a positive Wassermann with 0.2 cm. of fluid. With meningo-encephalitis Hauptmann and Nonne have shown that the fluid is negative when small quantities are used, but positive when 0.4 cm. are employed.

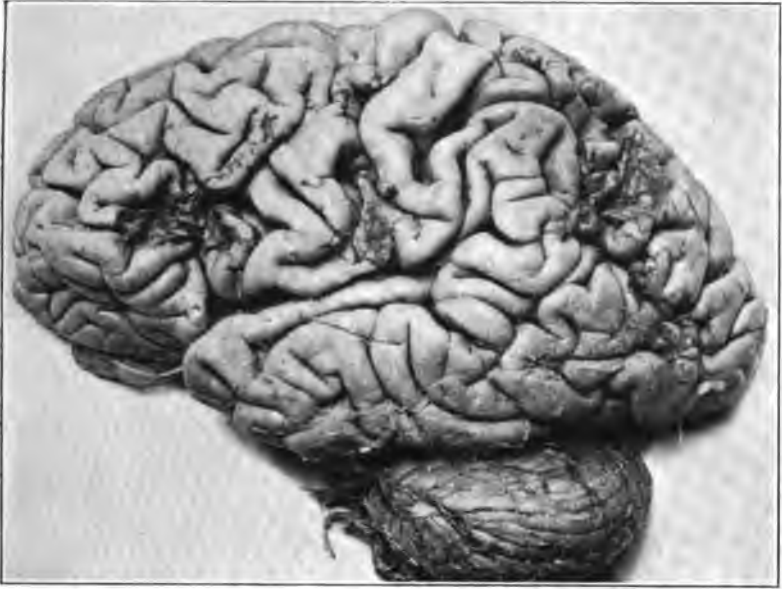
This generalization seems to hold true not only for convexity meningitis, but also for those in which the base is more especially involved, although, as will be later pointed out, in the discussion of paresis, variability in the Wassermann reaction is not unusual.

4. CEREBRAL SYPHILIS

Arterial Type.—In considering the symptomatology of patients who are thought to have cerebral syphilis, attention may again be called to the fact that the dividing lines between cerebral syphilis, basal meningeal syphilis, convexity syphilis, etc., are indefinite. Rarely does one find a pure basal or convexity meningitis without some involvement of the cerebral substance on the one hand, while it is probably as rare to find syphilitic processes strictly limited to the cerebral substance, and not involving the meninges. One can postulate pure types

PLATE XX

Fig. 1



Cerebral Syphilis of Convexity. Chronic Epileptiform Convulsions.

Fig. 2



Chronic Epileptiform Convulsions due to Syphilitic Leptomeningitis and Syphilitic Vascular Disease. (Lafora.)

PLATE XXI



Cerebral Syphilis (arterial type) with Softening. Aphasia, Hemiplegia, Advancing Dementia. (Lafora.)

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for purposes of description, but disease is rarely a respecter of one cerebral tissue more than another.

Symptoms.—It is for this reason that one is constantly reminded of the multiplicity of symptoms found in cerebral syphilis. In this connection it would not be without profit to glance for a moment at the diagnoses of certain cases reported by Nonne in his monograph several times alluded to. The patients were illustrations of basal, or convexity, or encephalic brain syphilis, usually combined forms. The short descriptive diagnoses run as follows: Specific headache a year after infection, with secondaries in skin and mucous membranes; headache and pupillary anomalies; headache and obstinate vomiting with tertiary testicle signs; progressive simple dementia cured by treatment; progressive dementia with defect; combination of convexity, meningitis and paresis; gummatous meningo-encephalitis of convexity with general symptoms, choked disk, and antisyphilitic treatment unavailing; surgical treatment of gummata, with cure; Jacksonian epilepsy; cortical epilepsy, choked disk, paresis of left leg; cortical epilepsy, optic neuritis, arterial hemiplegia; cortical hemiepilepsy and general cortical symptoms; arterial hemiapoplexy, with hemiepileptic convulsions; generalized epileptic seizures; hemianesthetic attacks, with cortical general signs; uremia, etc. Such illustrations might be almost indefinitely continued. They are not the exceptions, they are the rule. One is tempted to indulge in the generalization that one hundred consecutive patients with the types of cerebral syphilis under consideration would show one hundred different clinical syndromes. Practically all of those just enumerated belong to the convexity types of cerebral syphilis. Their enumeration may prove of service in localizing the process.

A similar series for the basal types may be equally of service: Gumma of right frontal lobe; anosmia; pressure neuritis of optic nerve; choked disk with general cerebral symptoms; bilateral neuritic optic atrophy; recurrent optic atrophy; hemianopsia; bitemporal hemianopsia; homonymous left-sided hemianopsia, with right-sided abducens palsy; temporal hemianopsia; cranial nerve and epilepsy; hemianopsia; hemianopic pupillary reaction, oculomotor palsy, epilepsy; ptosis; internal rectus palsy; optic atrophy; partial oculomotor palsy, fifth, seventh palsy; second, third, fourth, fifth, sixth nerve involvement; fifth, sixth, seventh, eighth nerve, right arm and leg palsy; seventh, eighth, psychosis (paranoia acuta), with manic moods; third, fourth, sixth, tenth, eleventh nerve palsies; second, third, fourth, sixth, seventh with epilepsy; third, fourth, seventh, polyuria; isolated internal ophthalmoplegia—to mention any more would be to needlessly extend this chapter. The lesson such findings indicate is obvious.

But eliminating, as far as possible, the varied syndromes of convexity or basal syphilitic meningitis, and limiting the discussion of the present section to those forms of cerebral syphilis due more particularly to arterial disease, what is its more frequent symptomatology?

In the first place it may be mentioned that arterial types of brain

syphilis may be found very shortly after infection—as short a time as two or three months. Naunyn in a thorough study found that 48 per cent. of 335 cases reported on by him developed signs of cerebral syphilis within three years. On the other hand, forty years have been known to elapse between infection and the development of a cerebral syphilis. There is no rule, and Fournier's large statistics are not borne out by other workers.

Here the prodromal symptoms are usually headache, dizziness, sleeplessness, irritability, inability to apply one's self continuously to one's work, lack of interest in work, etc.; in general, the so-called neurasthenic syndrome. These are naturally not absolute.

Headache.—The headache is usually very disagreeable; it usually has a migratory character—here, there, and elsewhere, usually dull, it is at times boring. It is inconstant, intermittent, often, not by any means always, worse at night than in the daytime. It may disappear for weeks or months, and then suddenly reappear. It may also be the only sign of cerebral syphilis for months or even years.

Dizziness.—Dizziness, in shorter or longer attacks, is very significant of the arterial disease. It is usually associated with the headache but may appear as the single symptom of brain syphilis. Like the headache it is apt to be increased by mental or physical work.

Insomnia.—Insomnia is frequent, often obstinate, quite variable and not infrequently sleep is made irregular and non-restful by the sense of heaviness in the head or actual headache.

Psychical Disturbances.—Psychical disturbances are the rule in these patients with arterial disease of the cerebrum. They become more or less apathetic, lose interest in their work; are unable to work because of forgetfulness or inefficiency. With this there is increasing irritability; an inability to size up the situation. Such severe disturbance is arrived at only after some time as a rule.

Abnormal Sleepiness.—Abnormal sleepiness, coming on in attacks, is not infrequent—such periods of torpor or apathy often intermitting with periods of anxiety or of acute restfulness or excitement. Periods of stupor or semidrunken states may occur. They often presage more distinct neurological signs, being based as they frequently are upon sudden extravasation, infiltration, or thrombotic plugging off of the blood from small areas of brain tissue.

Many patients with cerebral syphilis of this general character remain in this condition, it may be for some time; they show a picture precisely similar to certain patients with general paresis. It is in this general group that the greatest difficulties in diagnosis occur.

As has been previously stated a positive "four reactions" is the sole criterion for differentiating the two in the present state of our knowledge.

Brain tumor is also to be thought of in diagnosis. Paresis, brain tumor, possibly a gumma, cerebral syphilis, at times cannot be distinguished one from another clinically. With brain tumor, non-

complicated by syphilis, the absence of the four reactions affords a positive criterion.

Local Symptoms.—As a rule, however, the greater number of individuals with cerebral syphilis develop local symptoms, and neurological rather than psychiatric syndromes come into relief, or the latter are intermingled with the former. Palsies develop. These are transitory, partial not widespread, or may be severe, complete, and permanent, showing various hemiplegic syndromes, according to the anatomical site of major disturbance—usually thrombotic—cortical, capsular, midbrain, peduncles, pons, or medulla—the symptomatology of the different forms of which are dealt with in this volume in the chapters on Hemiplegia, Midbrain Syndromes, Aphasia, Apraxia, etc., by Drs. Tilney, Holmes, Russel, and Wilson.

Successive attacks of mild and transitory palsies are very significant of cerebral syphilis.

Monoplegias are not infrequent, one arm, one leg, one side of the face, possibly the cortical speech areas with, in case of double lesion, pseudo-bulbar palsy. Minor speech disturbances are extremely frequent, tremors of the facial muscles usually accompanying the stumbling, stuttering, or drawling speech.

A list of the usual clinical diagnoses similar to those already outlined for basal or convexity meningitis would show a multiplicity of phenomena, no less complex, in the neurological field and certainly infinitely more varied in the mental symptom pictures. As these latter will be discussed more in detail in the section on the Psychoses (p. 342), no further mention will be made of them here.

Details of Treatment of Cerebral Syphilis—This section will deal with the treatment of the forms of syphilis of the nervous system which have been discussed in the four preceding paragraphs. The treatment of other forms of nervous syphilis will be discussed in following paragraphs. The treatment of certain special syndromes, hemiplegia, aphasia, etc., for instance, which, while probably due to syphilitic brain disease as well as to other factors, is taken up in other chapters.

Insofar as they are conditioned by cerebral syphilis the treatment of the syphilis will be considered here, but the care of the after-results will be found in the chapters referred to.

As has already been noted, the general trends of the reaction to the syphilitic organism in the nervous system are toward (a) specific inflammatory changes in the membranes or bloodvessels leading to alterations of the blood supply through thromboses and, hence, to death and destruction of nervous tissues, or to new growths, gummata, which may remain or break down; (b) to secondary toxic changes, accompanied or not by the specific inflammatory changes just noted, and constituting the general reaction type termed para- or meta-syphilitic and (c) to such forms of modification of the germ plasm through toxin activities which results in impaired, debased, or neural development, with or without the presence of changes found in trends (a) or (b). These may be summarized under the head of hereditary syphilis.

In this section, treatment will be limited to the specific inflammatory changes just outlined. It is preventive and specific.

Prevention of Cerebral Syphilis.—The large problem of prevention of syphilis in general cannot be discussed here. In Volume I of this work, Havelock Ellis has taken up the problem of individual sexual hygiene and self-control. Until the human individual metamorphoses the superabundant sexual energy, which nature lavishly bestows upon him, into other forms of productivity than sexual intercourse, prostitution and syphilis will remain a menace. That powerful instrument for cultural advance, the brain, will continue to pay its toll so long as it falters in its task in properly controlling the race perpetuation impulse—confessedly the most imperious of all impulses of the human species. Complete release from syphilis will only come when this ideal has been attained. At the present stage of man's control or modification of the sexual impulse syphilis is one of the powerful allies of this ideal toward another type of sexual ethics. It is rarely known to be such, but this unconscious feeling is responsible for much of the attitude of enlightened humanity toward many of the sexual problems of the day. It is largely by reason of this that there is so much difference of opinion as to the best modes of legislation.

Any attempt to legislate the creative instinct out of man is doomed to disappointment. Education will bring about a better cultural standard, and syphilis itself will be an element in the educative process. Prostitution is only secondarily an economic problem. It is a fundamental situation which occupies a central position in sexual science, and which fortunately is being regarded in the light of a scientific rather than a religious problem. It will only be when the biological significance of the creative instinct is grasped, and social economic situations are adapted to meet it that such problems of responsibility and relative abstinence can be worked out. Then a scientific sexual ethics will arise which will not need syphilis or gonorrhea as one of its necessary co-workers. Our present sexual ethics is a survival of an older civilization. It was adapted to States when slavery was the normal situation, when women were regarded as slaves, as property; when women were undervalued; when individual love (romantic love) was deprecated; and when work was despised. The debasement of work leads to an aristocratic snobbery which is an important standard in the situation. Religious and legal ideals—also survivals—are founded on this slave ethics and are in need of serious revision if a newer cultural adaptation is to result.

Legislation thus far has made a farce of the whole business. What has been done helps to fatten the purses of corrupt politicians and other bits of the legal machinery. In fact, the prevalent mode of attacking the situation is by means of a hypocritical fostering of the most corrupt elements of society for personal exploitation—political and judicial. The subject cannot be further entered into here.

Individual prophylaxis is entitled to serious consideration. Army and navy regulations by the use of protectors, gray ointment or other

antiseptic to smear the prepuce after coitus, washing of the meatus with argyrol or similar preparation, these and similar procedures have cut down the amount of syphilis in the army and navy considerably. Such methods of general prophylaxis are better treated of in a work on sexual hygiene (Bloch).

The treatment of syphilis in its early stages is also not material for this section. Fournier's dogma: strike hard, quick and often, is the surest way of preventing nervous syphilis. Notwithstanding apparently thorough treatment in its early stages, secondary and tertiary syphilis of the nervous system appears.

Active Treatment.—The treatment of cerebral syphilis, appearing in any of the forms previously enumerated, is often extremely satisfactory. In fact at times one might say the results are often too good, as the speedy relief not infrequently leads the patient to forego further treatment, or causes him to pursue his course half-heartedly.

Cerebral syphilis for the most part is accompanied by active spirochetes; hence, the therapy is to be directed against this organism. For spirochetal poisons we possess mercury, arsenic, and iodine. Mercury and arsenic are active, iodine is very weakly toxic, but Neisser's most recent studies tend to show that it has actions other than that usually ascribed to it, namely, to promote the taking away of breaking down syphilitic tissue or its product—its so-called resorption action. Neisser teaches that iodides in large doses are toxic to spirochetes as well.

The treatment of nervous syphilis then should be an attempt to follow out a general antisyphilitic treatment, with special attention to certain structural peculiarities of the nervous system. These peculiarities are of much importance. In the first place, small lesions in the central nervous system, by impinging on important centres, bring about disastrous results, optic atrophy, pontine, medullary hemorrhages, etc. The enormous importance of correct nervous and mental functioning in the struggle for existence is self-evident. The difficulties in the way of repair are enormous—at times insuperable—in nervous tissues.

Hence, one's attack upon nervous syphilis should be prompt and complete. A complete sterilization is desirable—and this is often extremely difficult to bring about in nervous tissues.

It must always be borne in mind that cerebral syphilis is often present with a negative Wassermann—this finding then should not deter one if there are clinical signs of diagnostic import. Not infrequently active antisyphilitic treatment (salvarsan) causes the appearance of a positive blood reaction—how often this occurs is not yet known.

Study of the cerebrospinal fluid is often a better guide to the correct appreciation of the situation as well as a reflection of the activity of the process. Lymphocytosis is often present months or years before any definite nervous signs. Lumbar puncture is too often neglected in cerebral syphilis. Many authors state that if the blood is negative to Wassermann there is little occasion for studying the cerebrospinal

fluid. This leads to bad results. Vascular and meningo-vascular processes may progress for years in nervous tissues without giving rise to a positive Wassermann. Here is a situation in which this symptom fails to be present.

An energetic cure should therefore be carried out if there are suggestive signs of nervous syphilis, even should there be a negative Wassermann.

As to what an energetic cure should consist of there are divergent opinions, even among the most experienced. One reason for this is that syphilitic reactions in nervous structures are associated with special dangers, and hence, certain precautions are suggested. Not infrequently, as in the case of thrombosis, the syphilitic disease *per se* is not in nervous tissues at all, but in possibly a fairly distant blood-vessel. The syphilitic process about the bloodvessel can be modified; the degeneration in an area cut off by the thrombosis cannot be changed at least not by remedies directed toward the syphilis. Hence in nervous syphilis the problems of treatment are often of a special kind.

In certain patients, rapid action seems imperative; in others, the need for this rapidity is not so much in evidence. Granted a knowledge of these requirements, it is not as simple as it might seem as to the choice of remedies. Iodides given by the mouth in doses of 30 grains, 2 grams a day, show evidences of activity in about one week. Mercury by inunction shows results in about five days, while the newer arsenical preparations show reactive capacities in about forty-eight to seventy-two hours. None of these figures should be accepted as final, so far as curative action is concerned, nevertheless they are worth something. It is not apparent that iodides have a rapid toxic action; hence, in lesions which are characteristic of the more florid aspects of spirochetel growth—basal meningeal types of acute onset particularly—they should not be chosen in the initial attack.

Inunction Method.—For years neurologists have taught—chiefly under the influence of Erb—that nerve syphilis is best attacked by the inunction method—combined with iodides. In those situations in which the time element is of less moment this attitude seems justifiable.

Oleate of Mercury.—Different possibilities are here presented. The oleate of mercury is of value in that it is comparatively cleanly and produces results as rapidly as other mercurial preparations applied to the skin. A dram of the 10 per cent. oleate is to be used night and morning for four days. The patient then takes a vapor bath and the same dose is used once a day for four days more. If sponginess and soreness of the gums do not appear—with cleaned teeth and gums—the double dose may be continued; otherwise a single dose should be utilized. In using the oleate one usually employs a small piece of flannel in the rubbing—the first dose should be larger as the flannel absorbs it, and the same piece of flannel should be used continuously.

The oleate may irritate the skin, but as it is absorbed fairly well from all parts of the body one can shift around more readily with it

than with other mercurial ointments. One object of using the oleate is on the ground of secrecy. This has certain obvious advantages which may be further emphasized by the rubbing of the remedy directly over or in the neighborhood of the chief localization of the lesion when possible.

This line of treatment should continue at least six weeks; after the first week 10 grains (0.6 gram) of potassium iodide t. i. d. should be administered during the course of treatment. There is very little advantage in raising the amount of iodide above 60 grains (2 grams) a day. After six or eight weeks the treatment should be discontinued absolutely—to be renewed not later than three months after the termination of the last treatment. A third and fourth course is advisable, even imperative if a positive Wassermann is present in the blood or lymphocytes above 10 to the c.mm. are obtained from the cerebro-spinal fluid.

Unguentum Hydrargyri.—Unguentum Hydrargyri is much used and widely recommended. It has the disadvantage of being dirty and of attracting attention. The latter may be partly obviated by adding some non-stainable coloring matter, or some smelling compound such as balsam of Peru. Attention can thus be diverted from its characteristic color. The ointment is used in daily doses of from 3j (4 gram) to 3ij (8 gram), best rubbed in in the evening in a fairly definite manner, and in places where the skin is more permeable. One uses the inner surface of the arm and forearm for the first rubbings, covering them with bandages, then the inside of the groin, then the popliteal space, then the abdomen and back. The fifth or sixth day the patient omits his rubbing—takes a Turkish bath and then starts over the same course. This course is kept up for thirty doses.

Other Details.—The care of the skin and of the mouth is naturally to be kept in mind. The blood Wassermann should be tested at the end of the period, and if strongly positive, or if spinal puncture shows active lymphocytosis, or if clinical signs seem slow in responding, the inunctions should be continued at least two or three weeks longer.

Checking up by Wassermann and lumbar puncture three or four months later, or any increase in clinical signs should determine a repetition of the treatment along identical or more strenuous lines.

Iodides, 30 grains daily, are to be given throughout the course of the inunctions. All medication should cease at the end of the cure, unless there are definite indications for its continuance.

Other mercurial inunction masses may be used. Those of value are the hydrargyri vasenol, vasogen, mitin, resorbin, which have special indications which may render them particularly valuable.

Injection Treatment.—Injection treatment attempts an even more rapid and energetic attack upon the spirochete. Many battles have been fought among syphilographers as to the comparative merits of the insoluble or soluble salts. When so much diversity of opinion can be found, it usually indicates that the real differences are usually

minimal. Hence, ease of administration, safety, painlessness, etc., determine the choice of the remedy in each case.

Calomel, mercury salicylate, and thymol acetate are among the more favored insoluble salts. Calomel has occupied a high rank and can be utilized in the following forms:

	Grams.
R—Hydrargyri chloridi mitis	5.0
Sodii chloridi	5.0
Aq. dest.	50.0
Mucilago Arabici	2.5
R—Hydrargyri chloridi mitis	
Ol sesami, 10 per cent.	

Pravaz syringeful every second or third day, preferably into the muscles of the thigh or back, for 12 to 15 doses.

The hypodermic use of calomel is often accompanied by much pain. Abscess and necrosis is not uncommon, and lung emboli may occur—with care, however, calomel given by hypodermic is free from danger.

Creams of calomel, devised by Lambkin, have been extensively used, as they cause less pain and give rise to no complications if blood-vessels are avoided. These creams should be sterile. The formulæ of some in use are as follows:

	Grams.
R—Calomel	5.0
Creosote	20.0
Camphoric acid	20.0
Palmitin	100.0

Inject 10 m. of this cream once a week for one month, to be replaced by the following:

	Grams.
R—Hydrargyri (metallic)	10.0
Creosote	20.0
Camphoric acid	20.0
Palmitin	100.0

This is injected in doses of 10 m twice a week for three weeks.

After six doses have been given, no more doses for two months. Four injections of the metallic cream are then given at fortnightly intervals. Then a rest for four months. Then four injections as before, and a rest for six months. Then a repetition of four fortnightly doses—an interval of one month, and a final series of four metallic cream doses.

English syphilographers have found these creams admirable in army and navy work. They are adapted for early stages better than for nervous syphilis, but are worthy of more extended trial in nerve syphilis.

The use of the insoluble salts has the advantage of a much more prolonged action of the mercury. They also have the disadvantage—all mercurial salts share in this, however—of irritation of the kidneys. If albumin is found before the use of mercury one should look for a

sypilitic albuminuria. Tuberculosis, diabetes, alcoholism, marked cachexia are additional factors to be carefully dealt with. Gastro-intestinal disturbances are frequent, but it is doubtful whether mercury can cause a neuritis.

Thorough cleansing of the mouth and the use of a chlorate of potash mouth wash are imperative.

The soluble salts in use are very numerous. They include the sozoiodolate, bichloride, lactate, succinamide, biniodine, benzoate, and cyanide. Fournier lists about 30. They may be injected within the muscles or into the skin superficially. All are somewhat painful, and accidents are possible. In general the dosage is from $\frac{1}{4}$ to $\frac{1}{2}$ of a grain. The injections are given twice or three times a week.

GENERAL SCHEME OF INJECTION. The following general scheme is suggested:

1. The site usually chosen is the posterior third of the buttock, to avoid the sciatic nerve and vessels.

2. The injection should be made deeply into the muscle, using each buttock alternately.

3. The syringe and piston should be preferably of glass, easily sterilized, and the needle of platinum iridium, about $1\frac{1}{2}$ inches in length, and sterilized.

4. The skin should be washed with ether soap, washed with freshly boiled water, and swabbed over with an antiseptic solution.

5. After insertion of the needle, the piston should be slightly withdrawn, and if any blood appears the needle should be reinserted in order to avoid injection into a bloodvessel.

SOLUTIONS.—The injection of the solution free from air bubbles can then take place.

Various solutions are in use. Only a few can be mentioned here

R—Hydrargyri sozoiodolate	gm. 0'2	gr. iij
Sodii iodidi	gm. 0'3	gr. v
Aq. dest.	gm. 10'0	5 iiss

Dose—10 to 20 minims constitute the dosage.

R—Hydrargyri lactatis	gm. 0'2	gr. iij
Aq. dest.	c.c. 18'0	5 iv

Dose—10 to 25 minims.

R—Hydrargyri succinamidi	gm. 0'2	gr. iij
Aq. dest.	c.c. 10'0	5 iiss

Dose—10 to 25 minims.

R—Hydrarg. chloridi corrosivum	gm. 0'5	gr. viij
Sodii chloridi	gm. 3'0	gr. xlv
Aq. dest.	gm. 100'0	5 iij

Dose—1 to 2 c.c. daily or alternate days.

The use of corrosive sublimate—following Lewin—is usually very painful.

R—Hydrargyri cyanidi	gm. 1'0	gr. xv
Cocaine hydrochloridi	gm. 0'3	gr. v
Aq. dest.	ad gm. 100'0	5 iij

Dose—1 to 2 c.c.

A useful variant of this combines the cyanide with arsenic and strychnine, as follows:

R—Hydrargyri cyanidi,			
Strych. arsenatis	āā	gm. 0/6	gr. ix
Cocaine muriat.		gm. 0/3	gr. v
Aq. dest.		gm. 60/0	5 ij

Dose—5 to 10 minims every other day for 20 to 25 doses.

Cocaine may be added to any of the soluble salts. Its addition lessens the pain.

Fournier has always advocated the use of the biniodide dissolved either in sterilized oil or in water. It is, he claims, painless, sure, and free from dangers. In 2457 injections only 9 produced pain. Such results, however, obtain only when the physician is very careful. Careless use with the biniodides will produce all of the accidents, pain, abscess etc.

R—Hydrargyri biniodide			
Olive oil—sterilized	gm. 10/0		3 iiss

Dose—One Pravaz syringeful every other day.

R—Hydrargyri biniodide			
Sodii iodide	gm. 0/2		gr. iij
Aq. dest.	gm. 10/0		5 iiss

Dose—1 or 2 c.c. daily or on alternate days, with gradual elevation of the dose if stomatitis or gastro-intestinal signs are not in evidence. Twenty to twenty-five injections constitute a course of treatment.

R—Hydrargyri benzoatis			
Sod. chloridi	gm. 2/5		gr. xlv
Aq. dest.	gm. 120/0		5 iv

Dose—1 to 2 c.c. daily or on alternate days.

Combined arsenical and mercurial injections were very much in vogue before the introduction of the salvarsan preparations. One of the most popular of these has been the arsenical salicylate or enesol. This remedy has been used widely in nervous syphilis and often with surprisingly good results. Schaffer speaks very highly of it. Its action in tabes and paresis is discussed in the paragraphs relating to these syndromes.

The combined use of the cacodylates and of mercury has been observed to give good results. The early reports of optic nerve disease apparently following the use of the cacodylates served to force these salts into the background. Inasmuch as such optic nerve changes apparently occurred in other than syphilitic patients, it would not appear that they are to be interpreted as instances of those neuro-recidives which have been so actively discussed since salvarsan has been introduced.

Salvarsan and Neo-salvarsan.—The history of the introduction of this remedy into therapy, the outlines of its composition, and modes of administration, etc., are discussed in a separate chapter by Dr. Nichols.

It remains for us here to discuss its action on the types of nervous syphilis already outlined. Its use in tabes, paresis, hereditary syphilis, is taken up under their respective paragraphs.

Any attempt at an exhaustive summary of the various reports upon this remedy in the treatment of nervous syphilis would require a special volume. A simple enumeration of the bibliography alone—best obtained in brief in Lewandowsky's *Handbuch der Neurologie*; articles by Forster and Schaffer and others in Nonne's discussion referred to later would require dozens of pages. Only the present (1913) drift of opinion will here be expressed.

In those forms of nervous syphilis here under discussion, namely, the exudative, inflammatory, hyperplastic, gummatous, and arterial forms, salvarsan is by far the most efficient remedy that we possess at the present time. One form needs to be excepted, that of the large gummata, for which surgery alone is adequate.

It would also appear that much larger doses of salvarsan are required for nervous syphilis than were used in the earlier stages of its administration. Since the use of adequate dosage the so-called neurorecidives have almost entirely disappeared. Of this, later.

Finally, it is the present trend of opinion that mercury and salvarsan combined gives the best results. Whether or not arsenic and mercury, both active spirochetal drugs, supplement each other in this combined use is not certain, but the results obtained have in many instances been very satisfactory.

At the same time it needs to be observed that a number of patients have relapsed, and the final results of therapy in nervous syphilis have been far from being as hopeful as had appeared. Because this has been so is no reason why it should remain so. The most obvious reason that stands out in many of the recent discussions that have taken place relative to this point is that the patients have been insufficiently treated. Finally, salvarsan has not been long enough in use to warrant anything but as yet tentative conclusions.

With this short summary of conclusions, a few words may be said as to its application and dosage. Whether neosalvarsan is to replace salvarsan or not cannot yet be determined. The intravenous application of salvarsan is the best method of giving it. It should not be given unless the patient is under some sort of supervision—in a hospital or remaining in bed—and minute attention to the technique is absolutely necessary to avoid certain dangers. It is highly important that fresh distilled water be employed if salvarsan is to be used intravenously.

DISADVANTAGES AND ADVANTAGES.—The present situation shows certain disadvantages which need to be discussed.

1. *The Cost.*—This is an economic question and raises the point as to the relative advantages of specific therapy by salvarsan and by mercury. Salvarsan is a stronger spirochetal poison than mercury; this seems established. Whether it will act upon latent spirochetal deposits in out-of-the-way tissues better than mercury has not yet been proved—in fact the crucial experiment to prove such a thing has

not yet been possible. Attempts at getting at some light on this situation are being made with congenital syphilis, in conjugal syphilis, and under conditions where certain experimental values seem to be fairly constant. As yet such light has not broken.

The advantages seem to lie with salvarsan if rapidity of action is imperative—yet after all this is only a matter of a few days—still twenty-four hours under certain circumstances may be vastly important.

Again, one is justified in using it or recommending its use in those affections for which mercury has seemed to fail. These are discussed later under the head of tabes, paresis, etc.

Finally the use of salvarsan is sanctioned from the purely hypothetical side if economics do not enter into the question. Its use has already raised and helped to answer a flood of questions relative to the development and growth of the treponema, its toxins, the reaction of the cerebrospinal fluid, the activation of latent or dormant spirochetal deposits, the bearing of the Wassermann reaction on lipid substances and arsenical stimulus to the same—all of which are of very vital importance.

2. *Herxheimer Reaction.*—A second disadvantage is the Herxheimer reaction, and the question of relapse or neurorecidive. A whole set of problems was raised by an apparently new type of reaction which came into prominence soon after the use of salvarsan. The knowledge of these led Ehrlich, among the first, to feel that perhaps the use of salvarsan in nervous syphilis was not only not to be regarded as having any promise, but on the contrary might be dangerous. Fortunately, this feeling has been shown to have been overcautious.

Salvarsan is not dangerous to patients with nervous syphilis, but yet certain peculiarities develop which are worth while noting. Certain patients with cerebral syphilis reacted with sudden accession of mental and nervous signs. Headaches increased, excitement became pronounced—in some convulsions appeared, coma rapidly became grave, encephalopathies and cranial nerve palsies were apparently called into existence, or were made worse. A few paretics died suddenly. Tabetic pains were made worse, and gastric crises became more insistent. I omit the many signs in other organs, herpes, diarrhea, albuminuria, etc.

Previously these signs were all attributed to toxic action on nerve tissue—neurotropic. But with increasing experience it would appear that this is not the case—at least only under circumstances to be examined. While science is not in a position to definitely explain what happens, it is probable that the so-called Herxheimer reaction is one primarily due to syphilis itself. It is complex, and seems to show (a) death of the spirochetes with increase of toxin formation (seen possibly by the sudden reappearance of the Wassermann reaction), (b) an increase in the inflammatory protective reactivity (seen in increased lymphocytosis), (c) swelling of the areas involved by tissue edemas, bloodvessel changes, etc., and (d) possibly other factors as yet not grasped, among which perhaps direct toxic action is to be reckoned with.

This reaction is of short duration, as a rule, and appears in those patients who have had either moderate, single doses, or small, repeated, but insufficient doses; but above all, according to Ehrlich, in those patients treated under conditions of faulty technique.

Benario has studied these neurorecidives with great thoroughness, and although his position is possibly one-sided, yet it is worth considering here. Up to 1912 he had collected some 210 cases of cranial nerve neurorecidive. Sixty-five per cent. of these occurred in men, 35 per cent. in women—a proportion somewhat similar to that reported by Nonne among 185 cases. Six per cent. occurred in the primary stage of syphilis, 18 per cent. in the combined primary and secondary, and 75 per cent. in the secondary stages of the disease.

Of 190 cases the nerves were affected as shown in the table. At the same time, Benario reports 122 cases of neurorecidive under mercurial treatment.

Nerves involved.	Under salvarsan. Per cent.	Under mercury. Per cent.
Optic	29.1	25.1
Oculomotor	8.6	11.5
Trochlearis	2.3	—
Trigeminus	2.7	0.7
Abducens	5.9	2.3
Facial	15.3	23.0
Acoustic	35.0	35.8

A combined statistic shows the figures to be almost the same, tending to prove that the action is not neurotropic but syphilitic.

Later studies tend to show that these neurorecidives disappear entirely with a more intensive salvarsan therapy. The practical conclusion is that it is not proved—save for a few cases of neuritis—that salvarsan has done harm, and it has been shown that it has a very positive spirocheticidal action.

To kill spirochetes in the nervous system, however, is one thing, and to overcome the results of tissue changes is quite a different one, and this above all is the stumbling block in the treatment of nervous tissue syphilis. Nevertheless, if nerve tissues have not been extensively destroyed, one can hope for excellent results by a proper combination of salvarsan therapy with mercury.

Salvarsan must be used in much larger quantities, however, than was at first thought. In the section on tabes (p. 369) the outlines of an energetic combined therapy is given, and reference may be made to those pages for the general indications of such a course of treatment. Modifications to a less active mercurial salt than calomel may have to be made. Every patient needs individual treatment.

Notwithstanding the very evident fact that salvarsan and neo-salvarsan are active spirocheticidal drugs, it is still an important problem how to reach the nervous system with them. Careful chemical investigation of the cerebrospinal fluid has heretofore failed to obtain any trace of arsenic when salvarsan has been given in the usual manner. One may infer that the arsenic has become fixed in some chemical combi-

nation which fails to react to the usual chemical tests. It is not yet comprehensible why nervous syphilis is so resistant to treatment and why the hopes aroused by the striking results of salvarsan therapy in general syphilis seem not to have been borne out in nervous syphilis.

Intraspinal Therapy.—New methods have been ingeniously devised by Homer Swift and Arthur Ellis, working in the Rockefeller Institute Hospital. We shall have occasion to outline their technique in other details in the discussion in the sections on Tabes and Paresis, but an outline of the principle is given here with certain of the results obtained in cerebral syphilis by their method of application.

They have attempted to place a spirocheticidal solution directly into the cerebrospinal fluid. Salvarsan and neosalvarsan were employed by direct injection into the spinal canal through the Quincke lumbar puncture. This method they found was to be condemned. It failed to give any beneficial results, and, moreover, caused marked pains. It is probably a dangerous procedure, as animal experimentation has shown.

An attempt was then made to introduce into the patient's cerebrospinal fluid some of his own blood serum which had previously been mixed with the salvarsan by intravenous infusion. This infusion was carried out in the usual manner. After a certain length of time—one hour was found to give the most active serum—blood was withdrawn, separated from its corpuscles, after twenty-four hours, diluted to 40 per cent. with normal saline, and then heated to 56° C. for thirty minutes. A lumbar puncture is then made, and from 5 to 15 c.c. of cerebrospinal fluid is withdrawn, *i. e.*, until the pressure falls to 30 mm. of spinal fluid. 30 c.c. of the warmed serum are then slowly injected into the subarachnoid spaces. The patient must lie quiet, the foot of the bed usually being raised. After ten days to two weeks the injections, which are usually well borne, are to be repeated.

They thus obtained very striking results in the action upon the serobiological factors known to accompany cerebral syphilis. Then a more crucial experiment was planned. This consists in the introduction into the subarachnoid spaces of serum taken from another individual, usually a secondary syphilitic under treatment. The technique being that just outlined. In the charts here reproduced, and in the section on Tabes, those patients treated solely by spinous injections received their salvarsan solely from the serum of other patients. They received none directly. Hence the striking character of the results, for it is at once evident that the amount of spirocheticidal substance in a few cubic centimeters of serum taken from the body of another patient who had received the usual intravenous salvarsan therapy must be very small indeed, *i. e.*, reckoned as arsenic. If other factors than the salvarsan itself enter into the situation these are as yet unknown. One significant fact, however, would tend to indicate that other forces are operative. Extensive experiments carried on by Swift and Ellis with the heated and unheated serums show that the heated sera are three times as spirocheticidal to *Spirochæta duttonii* in mice.

History No. 605. F. K., aged twenty-seven years; cerebrospinal syphilis; duration unknown; primary unknown.

Date.	Blood.	Cerebrospinal fluid.				Treatment.		Clinical condition.
		Cells.	Noguchi globulin.	Wassermann reaction.		Intravenous.	Intraspinal.	
				Liver antigen.	Heart cholesterolin antigen.			
1912 Apr. 23	+ ±	60	++	0.2 c.c. ++	30 c.c. 40% serum 30 c.c. 40% serum	No symptoms; unilateral mydriasis the only physical sign.
Apr. 25, 26	59	+	0.2 c.c. ++	0.3 gm. salvarsan	30 c.c. 40% serum	
May 2, 3	++	0.3 c.c. ++	0.3 gm. salvarsan	30 c.c. 40% serum	
May 9	+	14	0.3 gm. salvarsan	30 c.c. 40% serum	
May 23, 24	+	14	±	0.3 c.c. ++	0.3 gm. salvarsan	30 c.c. 40% serum	
June 13, 14	+	5	±	0.3 c.c. ++	0.3 gm. salvarsan	30 c.c. 40% serum	
June 20, 21	+	6	±	0.4 c.c. ++	0.3 gm. salvarsan	30 c.c. 40% serum	
July 18, 19	±	2	±	0.5 c.c. ++	0.3 gm. salvarsan	30 c.c. 40% serum	
July 26	0.3 gm. salvarsan	30 c.c. 40% serum	
Aug. 15, 16	±	2	±	0.4 c.c. ++	0.3 gm. salvarsan	30 c.c. 40% serum	
Sept. 12, 13	±	6	±	0.4 c.c. ++	0.4 gm. salvarsan	30 c.c. 40% serum	
Oct. 3	±	0.3 gm. salvarsan	30 c.c. 40% serum	
Oct. 10	+	0.3 gm. salvarsan	30 c.c. 40% serum	
Oct. 17	—	0.3 gm. salvarsan	30 c.c. 40% serum	
Nov. 2	7	—	0.5 c.c. ++	0.6 gm. salvarsan	30 c.c. 40% serum	
Nov. 22	+	0.6 gm. salvarsan	30 c.c. 40% serum	
Dec. 12, 13	—	8	—	0.5 c.c. ++	0.9 gm. salvarsan 0.9 gm. neosalvarsan	30 c.c. 40% serum	
1913 Jan. 3, 4	Heart antigen. + ±	4	—	0.5 c.c. —	0.5 c.c. ++	0.9 gm. neosalvarsan	30 c.c. 40% serum	Unchanged.
Jan. 31, Feb. 1	++	3	—	0.5 c.c. —	0.5 c.c. ++	0.9 gm. neosalvarsan	30 c.c. 40% serum	

This would raise the question whether or no the heating alone caused alterations in the salvarsan serum which was advantageous in the therapy.

The most striking suggestive result is an almost immediate diminution in the number of pathological cells in the cerebrospinal fluid. The globulin reaction diminishes, positive Wassermanns of the cerebrospinal fluid with small quantities of fluid require larger quantities to show positive or become negative, and the amelioration of the symptoms has been rapid.

Through the courtesy of Drs. Swift and Ellis, I here reproduce a chart of a patient with cerebral syphilis, which shows these features. Others will be found in the sections on Paresis, Tabes, Meningomyelitis, and Radiculitis.

The Table on page 339 is to be interpreted as a result of combined intravenous and intraspinal medication. The cell count, Noguchi and fluid Wassermann, show marked changes indicating a diminution in the syphilitic reaction.

Mercury by the Mouth.—Mercury by the mouth will always remain one of the simplest, and yet, at the same time, least efficient methods of treating syphilis of the nervous system. Here again one has a rich choice of remedies. Those most in use are the protiodide, grain $\frac{1}{4}$ to $\frac{1}{2}$ sublimate grain $\frac{1}{10}$, calomel 2 to 5 grains. Various vehicles are used.

The disadvantages of treatment by means of the intestinal canal are many—chief of which is the slow and weak action of the remedies employed. Moreover, the gastro-intestinal tract suffers.

The chief advantage is that mercury may be combined with the iodides. Furthermore, conveniences of medication must often constrain one to use this mode of giving antisymphilitic remedies.

In nervous syphilis it would appear that mercury medication by mouth is not radical enough. One may use it after an energetic cure, by the methods outlined, has been employed, but oral administration is rarely a method of certain value, and hence is not advisable, save under particular circumstances.

Among the newer mercury preparations which future experience may prove to be of value are: Mercury dicarboxylate, two of which are on the market, with pronounced toxic action on spirochetes in rabbits. It is claimed to be twenty times as toxic to spirochetes as corrosive sublimate, and yet shows no action on the body. Its dosage has not yet been worked out.

Mercury glidine (luesan) is a combination of mercury with lecithin and albumin. It is given in tablets of 0.005 to 0.01 gram ($\frac{1}{17}$ to $\frac{1}{8}$ grain), 3 to 6 a day.

Mercury guaiacol sulphonate is used for intramuscular injection in doses of 0.02 gram ($\frac{1}{8}$ grain) in sterile water on alternate days for 10 to 20 injections.

Mergal is used by mouth in capsules, and has been found to affect both primary and secondary syphilis. Its action on nervous syphilis has not yet been widely reported on.

Merjodin is given in tablet form in increasing doses until 9 to 12 tablets a day are being taken. Diarrheas are not infrequent side effects.

Iodides.—Sodium and potassium iodide have been used in the treatment of syphilis of the nervous system for years, and often with good results. According to Neisser the iodides are weak spirochetal poisons. Our belief in its resorptive powers is justified on empirical if not on pharmacological grounds. Personal experience does not confirm the belief in the efficiency of especially large doses, although that is the American preference.

The use of the iodides in doses of from 10 to 30 grains t. i. d. combined with mercury is particularly valuable in the gummatous type of cerebral syphilis. It is folly, however, to try to do away with large gummata by means of massive doses of iodides.

The dosage of the iodides will depend upon the individual. There are many idiosyncrasies to be borne in mind. At times small doses cause marked disturbances and cannot be borne. Here one may employ other combinations than those of sodium or potassium. Hence, strontium, rubidium, and organic iodine preparations have come into use. Iodopin, sayodin, iodoglidin, iodoval, iodocitin, iodostarin are among the newer of these combinations.

Iodopin may be injected as well as administered by the mouth. In the former case it is used in quantities of 10 c.c. on alternate days, or smaller doses 1 to 3 c.c. at more frequent intervals. In giving it by hypodermic, both the syringe and the remedy should be slightly warmed, the needle should have an ample bore, and the drug be introduced slowly. It is also given by the mouth in 3j doses. In the form of iodopin, large quantities of iodine may be introduced without toxic effect. Its action on nervous syphilis has not been extensively studied. Good results are reported by its use in syphilitic labyrinthitis.

Iodoval, and iodocitin, the latter a lecithin albumin compound, have been found to be borne well in the course of salvarsan-mercurial treatment. The former is given in doses of about 5 grains t. i. d. throughout an energetic salvarsan-mercury cure—the latter in about the same doses.

Iodostarin is given in tablets of 4 to 8 grains t. i. d. It is a compound of taurinic acid, insoluble in water. The iodine ion is split off supposedly in the small intestine, and iodine is slowly eliminated, seeming to show a more prolonged action in the body than other iodine compounds. It has been found of value in headaches of nervous syphilis, but its use is still uncontrolled by much experience in nervous syphilis.

The whole *modus operandi* of iodine medication in syphilis is in need of more understanding. Empirically it is known to have value—but why is not yet thoroughly grasped.

Surgical Treatment.—One here considers brain puncture, callosal puncture, trephining and cranial explorations for gummata or gummatous meningitis.

Certain patients with evident signs of internal or external hydrocephalus due to syphilis and whose symptoms do not seem to be influenced by antisymphilitic treatment may be benefited by lumbar puncture or by trephining, or by a puncture of the ventricles or even a puncture of the corpus callosum. Here headaches, spasticity, and signs of choked disk are the indications following an energetic drug therapy with negative or minimal results.

Large gummata may be handled, like other forms of brain tumor, by operation. Horsley, Starr, and others have reported some excellent results. Surgery not only removes an offending mass which is rarely broken down by antisymphilitic remedies, but also avoids the sclerotic scars of those that might be reduced by such therapy.

With the steadily improving technique of cranial surgery, there seems little to contraindicate handling a cerebral gumma, that can be fairly accurately localized, on purely surgical grounds.

If six to eight weeks of energetic specific treatment fails to diminish the symptoms; if a persistent, if stationary, syndrome exists; if a Jacksonian epilepsy persists, even after the regression of general tumor signs, then operation is to be recommended.

General Management of Nervous Syphilis.—This will be found in the section on the treatment of the spinal form of nervous syphilis, as many bedridden cases belong to that group. (See p. 408.)

5. GENERAL PARESIS: ADULT AND JUVENILE

It is usual to separate paresis as well as tabes from other syphilitic disorders of the nervous system, under the general caption of para- or metasyphilitic disorders. Fournier is largely responsible for this, and to paresis and tabes he has added a number of other disorders, in other parts of the body, to which he applies the term para- or metasyphilitic.

Just what para- or metasyphilis is is not known, especially so far as the nervous system—to which this chapter is limited—is concerned.

Many ingenious hypotheses have been formulated with the purpose of explaining the differences between paresis and tabes, on the one hand, and other forms of nervous syphilis, secondary or tertiary, on the other. It would serve little purpose to enumerate them in detail, since none has as yet compelled conviction.

To return to the syphilitic etiology of paresis. It is practically conceded "no syphilis, no paresis." One is not speaking now of those few individuals who, either because of the presence of a brain tumor, or the existence of arteriosclerosis, or of other cerebral disorder, show close clinical resemblance to paresis.

It may here be recalled that it was in 1857 that the causal relationship of paresis to syphilis was maintained by Esmarch and Jessen; since which time the battle of differing opinions has been unremitting. The appeal to statistics left individual predilection to interpret them as

PLATE XXII



Treponema pallidum in the Brain of a Paretic. (Moore.)

W. B. Moore

Arto

one wished. Statistics can neither prove nor disprove the thesis. The work of Wassermann and Plaut has raised the dictum here quoted: "no syphilis, no paresis," much nearer to a finality, while from the side light of comparative pathology the work of Spielmeyer and Mott on sleeping sickness, on horse syphilis, and diseases due to agents closely allied to the syphilitic organism has shown precisely similar pictures to those seen in paresis. The variants are not worth further discussion in view of the availability of Mott's writings to English reading students.

Finally the findings by Moore and Noguchi of *Treponema pallidum* in 12 of 70 paretic brains serve to render more certain the relationship of the organism to the disease. (Plate XXII.)

But syphilis does not by any means necessarily lead to paresis, fortunately. The most recent studies of Mettler show that about 2 per cent. of those infected by syphilis develop paresis. A considerably larger percentage develop cerebrospinal syphilis—how large one can only surmise.

The question then arises—how is it that in certain cases a disorder arises, usually more than five years after infection, which while closely resembling many forms of cerebral syphilis, yet differs from it in certain very noteworthy particulars, and what underlies these differences? In other words—why para- or metasyphilis?

Naturally there are those who say there is no difference, either anatomically, biologically, or therapeutically. They are in the minority with certain well-developed arguments, some of which are as yet unanswerable. The present-day attitude is to maintain a distinction between the strictly cerebral syphilitic disorders and general paresis, chiefly because the histological pathology is unique, the biological tests are different, and the results of therapy diverse.

Alzheimer and Nissl have laid down fundamental distinctions in the pathological picture. The chief points are quantitative, and to a lesser extent qualitative. Their researches have remained uncontroverted, although modified.

Wassermann and Plaut, by means of the biological reactions, demonstrated a further variation. This feature has been discussed already under the caption of the four reactions. Recent studies with large amounts of cerebrospinal fluid, as outlined by Hauptmann, and actively followed out now by scores of workers, have seemed to show that the specific differences claimed by Plaut are less definite than he supposed; but, as yet, no one is in a position to interpret the peculiar results obtained by increasing quantities of cerebrospinal fluid—results which are somewhat confusing and upsetting.

Finally the therapeutic test. This was the stumbling block for the earlier clinicians. The general inefficiency of treatment argued for the non-syphilitic nature of the disorder; but now that the relationship between *Treponema pallidum* and paresis seems certain, the failure to respond to treatment remains an enigma, especially when one bears in mind the surprising remissions which take place in this disease.

It is not without significance that a number of observers have claimed that the therapeutic test even is not decisive. Leredde (1903), in his extremely suggestive monograph, argues for the curability of paresis and tabes, especially if the therapy is begun early, and carried on energetically. While this position has not been entirely abandoned, the studies of later years, which utilizing the four reactions make more searching diagnostic criteria necessary, point to the fact that paresis is most resistant to antisyphilitic therapy. Whether such treatment is able to inhibit the development of paresis will be discussed later.

Symptoms.—The full symptomatology of general paresis cannot be taken up here. The syndrome is characterized by a bewildering multiplicity of forms, which, shifting in the individual patient from month to month, at times even from day to day, prevent any clean-cut description that will embrace its many vagaries. Attempts have been made to create empirical types. Thus, Binswanger, in a notable study, created three types based on certain anatomical peculiarities—meningitic-hydrocephalic, hemorrhagic, and taboparetic forms. Such an anatomical subdivision has no reliable clinical counterparts.

Attempts at the erection of clinical types are perhaps slightly more encouraging, for there are certain patients who will run a course true to type.

The earlier monographs of Simon and Kraftt-Ebing, and the later ones of Joffroy, Obersteiner, Klippel, and Kraepelin suggest the following groups:

1. Simple dementing types.
2. Simple depressed types.
3. The expansive or so-called classical type.
4. The agitated types.
5. The irregular types with localized symptoms, Lissauer, taboparetic form.
6. Juvenile paresis.

Before even attempting a description of these purely artificial creations, pictures which are constantly shifting and showing combinations of details, a brief glance at the chief symptom components is advisable.

These have frequently been divided into the mental and physical, but as this is a purely arbitrary distinction we do not purpose following it. As has been noted, a diagnosis of an impending paresis may be made, at times some years before its onset, by the findings in the cerebrospinal fluid, but our attention is here first focussed upon the mental picture. A peculiar psychical weakness is one of the early phenomena. A difficulty in perceiving external impressions shows this intellectual loss. In the early stages it may require special study of reaction times, which are usually lengthened, but soon absent-mindedness, inattention, loss of details, forgetfulness of important facts, becomes apparent. There is a gradually developing loss of ability for prolonged mental effort; in conversation finer shades of meaning are lost, the patient is no longer alert and keen, as perhaps has been his normal habit. The

intellectual deterioration going on leads to many changes in his usual conduct, until the patient may be no longer quite sure of himself in his customary surroundings.

Certain patients develop a state of dreamy consciousness, as though in a mildly intoxicated state.

Increased fatigability is another early symptom. Much has been written of the preneurasthenic stages of paresis. This excessive fatigue may prevent him from starting anything new—sometimes he even falls asleep while at work or in conversation.

Defects of Retention and Memory.—Retention and memory soon commence to show defects. Careful studies in the early stages have shown difficulties in association, lessened capacity for learning, disturbance in attention, often with good retention. The patients forget recent happenings more readily, not knowing what has transpired a week ago, yesterday, sometimes a half-hour ago. These grosser defects belong to the later stages as a rule. The memory of time relations gradually slips away, the patient being unable to arrange successive phases in an orderly series. Thus, many of these patients show the greatest defects in their appreciation of time differences, when married, age of oldest child, and related striking facts of life. In later stages all sense of time becomes effaced.

Impoverishment of Ideas.—A gradual impoverishment of ideas takes place, varying in degree from month to month and showing also great variability with different patients. Those associations most in use usually persist the longest, and all tests should take into consideration special aptitudes. In the later stages the patients have lost practically all of their mental possessions—the gap at times being filled in by retrospective, confabulatory reminiscences. Certain of these patients resemble patients with a Korsakow syndrome.

Loss of Judgment.—Loss of judgment naturally goes on *pari passu* with the general psychical disintegration. In the early stages even, refined methods of testing, such as those of Gregor, show that uncertainties, contradictions, logical lapses are not infrequent. The patients are easily distracted by sound associations $9 \times 9 = 99$, etc. As this loss of judgment goes on the patients may make the most absurd plans. They do the most unusual things, often involving their entire fortunes and playing havoc with all of their carefully raised social fabric. Dream world and real world become hopelessly confused in this fundamental intellectual crumbling.

Hallucinations, illusions, and changes in simple sensory perception are found, but they are not, as a rule, prominent features in paresis. Delusion formation is naturally present in many instances, although certain patients may go through the disorder with but few delusional developments.

Delusions.—The delusions vary immensely—they are usually senseless and fantastic and when combined with active imaginative factors as they frequently are, especially in agitated or excited periods, pass all bounds. These patients think in millions, billions, quadrillions,

etc. They are princes, kings, emperors, potentates, priests, Christ, God, Super Gods. They have rubies, pearls, diamonds, emeralds; two wives, a dozen, a harem, thousands of beautiful women, etc.

These delusions, simple or fantastic, are also liable to great lability. They are always changing; contradictory as well as unconscious. New ones come, old ones go, revivals take place. Progressions may go backward: now they have millions, next moment have thousands; now a king, in ten minutes a fine soldier. One can at times, by talking with these patients, expand or contract their delusional exuberance almost at will.

Alteration of Emotional Activity.—The disposition or emotional reactivity is involved, as is the intelligence. As a rule the patients in the early stages are hyperexcitable—others, however, are markedly depressed. They are apt to be touchy, surly, cross, even having violent outbursts for the most trivial events. There is often a distinct damper in their higher ethical feeling, so that the stimulus of conversation, the joy of music or art, of various social relations gives way to a careless indifference, often at great variance with the psychomotor activity of the patient.

As the disorder progresses the mood is apt to be colored by the delusional interpretations. Anger and laughter may follow one another in quick succession, and a great variety of fleeting, changeable, often contradictory emotional states are passed through.

Character Alterations.—The character alterations are predominant. Will power is progressively lost; instability and foolhardiness alternating with obstinacy and perverseness. All initiative is reduced, and the patient may become as clay in the potter's hand; such periods alternate irregularly with impulsive heedlessness. Kraepelin relates the case of a patient who stepped out of a second-story window to pick up a cigar that he happened to notice on the walk beneath him. Criminal actions may be committed in just the same manner as in the case of the paretic who shot at Mayor Gaynor, of New York. Suicide may occasionally take place in the same manner. Stealing is by no means infrequent, and sexual misdemeanors and crimes are extremely prevalent. This blunting of the repressions inculcated by the force of civilization is particularly noticeable, and predominantly in the sexual sphere. Hence results the frequent telling of lewd stories, frequenting with people of quite inferior social status, exhibitionism, shameful and open masturbation, and even sexual assaults.

Neurological Signs.—Here one finds not infrequently in the beginning phases a dull heavy headache. Hyperacusis often precedes the blunting of the special senses, and various localized disturbances, such as word blindness, word deafness, auditory hallucinations, apraxia, asymbolia, astereognosis indicate a special localization for the time being in more or less definite cortical areas. Optic nerve atrophy occurs, at times early, in from 5 to 10 per cent. of the cases. Special changes in the optic disk are recognizable in from 12 to 80 per cent. of the cases (Joffroy, Raviart).

PLATE XXIII

Fig. 1



**Paresis with Korsakow Syndrome; Wernicke's
Polioencephalitis Superior.**

Fig. 2



Set of Pictures of Parietic Convulsion. (Kraepelin.)

Fig. 3



**Paresis with Degeneration of Pyramidal Tracts, with
Spastic Signs.**

Perversion of Cutaneous Sensibility.—Very frequently cutaneous sensibility is perverted—sharp pain, numbness, itching, etc., occur, and in those forms recognized as taboparetic these often show the special localizations of the tabetic. Out of these perverted sensations delusional interpretations frequently arise. A general insensibility to peripheral stimuli develops later, and the patient may then pay little attention to any kind of irritant, heat or cold, full bladder, distended rectum, etc. Occasionally such patients mutilate themselves, cutting off a finger, or the tongue, or the testes in order to get rid of what seems to them a foreign body.

Motor Incoördinations.—Motor incoördinations, from initial trembling to more high grade ataxias, apraxias, adiachokinesias, Romberg, asynergias, are common. Intention tremor is not infrequent, and perseveration is almost never missed in the later stages.

Disorders in Speech.—In speech these motor difficulties have been specially studied since Esquirol first laid stress upon such changes in mental cases. Frequently beginning with slight stumbling, a slurring over certain letters or syllables, r's, l's, etc., the paretic develops very characteristic speech anomalies. These come out with marked prominence in the use of test phrases—electricity, Methodist Episcopal, organization, truly rural, third cavalry brigade, etc.—when certain letters are repeated, stumbled over, or elided. Paraphasia, aphasia, ataxia of speech, perseveration are among the frequent later developments until only a mumbling may be possible in some of the last stages. Similar changes take place in writing.

Eye Symptoms.—In the ocular movements, analogous difficulties are observed and in the pupils one observes a variety of changes, already discussed in the paragraph on diagnosis. Statistical studies show these pupillary anomalies to be extremely frequent. Differences in size from 50 to 80 per cent. (Räcke), distortion of the pupillary outlines (74 per cent. Joffroy), Argyll-Robertson pupil 50 to 70 per cent. (Westphal, Junius, Arndt, etc.), many of these pupillary anomalies undergo considerable variation, changing from time to time even without treatment. Loss of consensual light reflex, as already noted, is often one of the earliest, and at the same time one of the most persistent of these pupillary anomalies. Weiler has devoted special attention to these in an important monograph. Occasionally there is a reversal of the ordinary Argyll-Robertson phenomenon—a loss of accommodation reflex with preservation of the light reflexes.

Convulsive Phenomena.—Convulsive phenomena are rarely missed in paresis. They are usually of the cortical epileptic type. They often occur early in the disease or may punctuate any period in its development. At times limited, they more often are generalized, and frequently have prodromata, such as dreamy states, motor incoördination, thickness of speech, twitchings, etc., as a rule occurring early. As isolated phenomena, typical status attacks may be observed, with as many as 100 or more epileptiform crises in twenty-four hours. An attack in the very early stages may last only a few seconds; the patient

suddenly sinks back on his chair, and be all right in a few moments—while, on the other hand, status attacks may persist a week or even more. Unconsciousness is usual, though it may be very slight or fugacious. A vast variety of focal residuals have been described.

Similar changes may be observed on the sensory side of the nervous system, and so-called psychic equivalents, as in the more classical epilepsies, are frequent.

Statistical studies show the very great frequency of these attacks, Obersteiner recording them as often as in 90 per cent. of his patients; while Junius and Arndt in their recent extensive study give them as occurring in 53 per cent. A personal study of two hundred cases showed them in 78 per cent. of the patients. Kraepelin believes that treatment in bed limits the number and frequency of the attacks; his Munich statistics show an incidence of about 65 per cent.

Alteration in Reflexes.—The tendon reflexes—triceps, radius, knee-jerks and Achilles—are usually positively involved, either as excessive, in the greater number of cases, or as diminished, especially in those patients with posterior cord involvement, which is frequent. When the deep reflexes are found to be increased other symptoms of involvement of the pyramidal tracts are not infrequent. Babinski reflex, very frequently Chaddock's external malleolar sign, at times the paradoxical reflex of Gordon occurs. Possibly there is an ankle clonus, and spasticity in gait is present. If, on the other hand, the deep reflexes are diminished, other signs of involvement of the position sense and deep sensibility fibers, travelling the posterior column pathways, are usually found. Ataxia, Romberg, girdle sensations, anesthiæ, etc., pains of the radicular type, are also often encountered in these taboparetics.

In most of the patients there is great variability in the two sides. Occasionally one finds spasticity of one side and hypotonia and ataxia of the other, and combined symptoms are to be expected in the later stages, especially in those patients with prominent cord localizations.

In the final stages contractures occur in the bedridden patients. They are unable to do anything and muscular twitches, spasms, localized atrophies, and a veritable museum of anomalies is to be looked for.

Findings in Cerebrospinal Fluid.—The findings in the cerebrospinal fluid have already been discussed in a preceding paragraph. (See Figs. 17 and 18, p. 312.) Suffice it here to say that they are of paramount importance and a diagnosis of paresis without the signs obtainable in the cerebrospinal fluid must always be regarded as lacking in a most important measure.

Enough has been said to show that the clinical picture of paresis may be closely counterfeited by a number of other pathological states—notably brain tumor, cerebrospinal syphilis, arteriosclerosis, chronic alcoholism, sleeping sickness, etc.

The findings in the fluid are usually definite. A positive four reactions, the fluid used in small quantities—0.05 to 0.2 c.c.—is almost certainly diagnostic of paresis, yet at times it would appear that

PLATE XXIV

Fig. 1



Paresis. Cortical Changes. Epileptiform Convulsions.
(Lafora.)

Fig. 2



Contracted State Paresis. (Kraepelin.)

1701



Group of Paretics. (Kraepelin.)

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positive four reactions are found in other syphilitic processes which do not behave like paresis. The earlier didactic attitude of Plaut seems to be in need of some revision, but at the present time there is not sufficient autopsy-controlled material to permit absolute dicta.

Vasomotor and Trophic Disturbances.—Vasomotor and trophic disturbances may appear early and come and go, among them skin eruptions, such as herpes, and pemphigus are the commoner types met with; the ready appearance of bed-sores and abscesses indicate the lowered resistance of the skin and subcutaneous structures.

The bodily temperature shows considerable variation, even on opposite sides of the body; it is usually subnormal, save following a convulsive seizure. Sleep is irregular, especially in the excited stages, when the paretic may not sleep for days—whereas, in torpid stages or in those quiet dementing forms the patient sleeps or is in a doze most of the time.

The appetite is capricious, and the bodily weight is apt to fall off in the early stages and during excitement, to become much increased in the torpid quiet states.

Disorders of the bladder, sexual organs, and intestines, all sooner or later come within the outlines of the picture.

Remissions.—One clinical feature which is very striking is the tendency of this disorder to show marked remissions. In certain respects this is a general law in disease processes, but in paresis it appears most striking because of the almost miraculous change that takes place in the patient. Such patients one would say were about to die; they become absolutely helpless, convulsion follows convulsion; in the interim they know nothing, are bedridden, soil themselves, and are reduced simply to breathing, heart-beating automata. They may remain in this condition for weeks and months, and then pick up a little, and then more and more and within a space of six weeks to three months many such patients appear to be almost well and like themselves. They have risen from the dead, and strange to say, although the relatives, friends, and business associates have been told over and over again perhaps, for they should be, that this is not a cure, that it is only a remission of symptoms, the patient is frequently restored to all his civil rights and given full control of his affairs. In the majority of cases this is disastrous; he may launch out into new lines, involve his fortune, marry unwisely, and then after a few months, perhaps a year—the longer remissions on record have been five or six years—the average is about six months—the symptoms return, often in rapid progression, and usually lead to death after variable intervals of from six months to a few years.

Forms.—To return now to the subject of the forms—those more or less artificial groups which for the purposes of description psychiatrists are agreed upon.

1. **Dementing Form.**—That which characterizes this general group is the progressive mental deterioration with motor paresis. Excitements, convulsions, extravagant delusion formations are not prominent

and when present are transitory. In these individuals there is the early period of nervous irritable weakness, with loss of mental alertness, moodiness, inability to work, forgetfulness, and steadily increasing poverty of thought. Naturally, the picture beginning in this way may suddenly change. This is sufficient to throw the patient into another group—but if the development is of the slow progressive nature, gradually advancing mental weakness, fleeting delusional ideas, often with childish, weak-minded features, these are the general symptoms of the dementing type of paresis.

2. **Depressed Forms.**—Here anxious depression is in the foreground of the mental picture. Hypochondriacal, delusional states are prominent. The patients continually complain about bodily discomfort; have lost their intestines, or have destroyed their manhood by masturbation or sexual excesses. These delusional ideas become more and more nonsensical. In many instances the hypochondriacal ideas are dependent upon fancied sinful actions or wrong-doing. They are great sinners, they must be protected from the police; fear they will be sent away. Such patients often have persecutory ideas, and when such are prominent early in the disease, before there is marked deterioration, they are frequently regarded as “paranoiacs” especially by those schools which regard names as disease entities and seek for diagnoses from a “pathognomonic” symptom. These paretics with persecutory ideas often have pronounced hallucinations of hearing.

Notwithstanding these hypochondriacal or persecutory, delusional interpretations, these patients are markedly indifferent: they are apathetic, talk, and move in a monotonous, dull manner, and take little interest in their surroundings.

Thus the loss of energy, the libido in Jung's sense, not being able to go forward in the affairs of life, a marked pathological introversion takes place, and the regression takes hold of all sorts of nonsensical, childish, infantile, and archaic phantasies. Pathological projection also is common, and one has a regular chaos of pathological mental mechanisms. The sense of reality is so markedly impaired, and the affective relativity so cut off, that the nonsensical beliefs have no corresponding or adequate emotional relationships. The personality is fragmenting and disintegrating.

Childish regressions of hero formation appear. The patient is a God, a king, an emperor; like Jack and the beanstalk, he is miles high; as in the Lilliputian, he is a great giant; as in Midas' touch, or Aladdin's lamp, he breaks the bank at Monte Carlo, or is the owner of immense gold mines, fabulously valuable jewels, etc.

Contrasting states of great inferiority, weakness, poverty, cause them to be very fearful, easily confused, easily lost; they beg for protection, hide from anger, or ask piteously for food, preserve scraps, etc.

They become unmanageable in bed, and finally in many the agitation and fear develop great resistance and violence. Self-destruction may be attempted, mutilation occasionally occurs. Most of these attempts, however, are fragmentary, non-sustained and bungling.

PLATE XXVI



Excited Paretic. (Kraepelin.)

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Stuporous states show a contrast to this marked violence. They may persist for weeks, months, or even years. The patients lie stupidly, "depressed," or anxious in bed, unclean and unmanageable. Special rigidities, catatonic like in their nature, may develop.

The special statistics show that from 15 per cent. to 20 per cent. of the material in some of the larger European hospitals and clinics may be in general thrown into this depressed category.

3. Expansive Types.—This general type has been for years considered "classical," yet they are not as frequent as the demented types. Thus, Kraepelin gives 30 per cent. in his Heidelberg series, Junius and Arndt 27 per cent. in their Berlin material. It is to be regretted that the specialist has failed to emphasize this feature, which is of so much value to the general practitioner, and has attempted to excuse his faults of observation behind a pseudoscientific discussion regarding a "change in type." Practically it is of more value to insist upon the comparative rarity of the megalomaniac features of paresis, since, as the average medical man has been taught to recognize paresis by this sign, it is not to be wondered at that so much delay has occurred before the recognition of paresis. The emphasis should then be laid not upon striking symptoms, but upon the apparently obscure ones.

These striking megalomaniac features are too well known to be dealt with here at any length—this is not a text-book on psychiatry. Here the boastful ego rises to superior heights. Everything is seen from the standpoint of an abundant energy. At first the ideas are those of great exaltation, within the bounds of normal human experience, but soon the patient loses his earthly bonds and soars to superhuman unrealities. His strength is appalling; his education superior to any others in the world; he speaks ten, nay, all languages; has all wealth; all power; figures mount from thousands to millions, to pages of ciphers. And in kaleidoscopic changes, and great individual variation one learns of many marvels of superior excellence only dreamed of in childish phantasy, or seen in the boasts of inferior peoples.

One feature of this frightful megalomania, which has its very great ups and downs, should never be overlooked; namely—the tendency for such patients to commit sexual indiscretions, even atrocities; or to engage in the most foolhardy enterprises, thus jeopardizing life and property.

This feature in paresis is of so much importance that a special paragraph will be devoted to the legal measures which should be invoked to prevent the worst consequences of this mental weakness.

That the megalomania has a distinct deterioration background is seen in the frequent combination of a poor clerk, in a state institution, who speaks of the million dollar novel he is writing. It consists of a few miserable scrawls on toilet paper, or on the edges of a daily newspaper. This is only a type. Such inconsistencies may be read of in the classics of psychiatry, from the work of Arnold to the present time.

These fantastic, exalted, euphoric states very frequently elaborate on sexual themes. Thus the patients have hundreds, millions of

wives or concubines—"Solomon was a piker in this matter" boasted a Bellevue patient. The children are more numerous and more beautiful than any promised to the ancient Hebrew heroes.

One patient, mentioned by Kraepelin, could lift ten elephants, is two hundred years old, 9 feet tall, is a beautiful Adonis, weighs four hundred pounds, has an iron chest, an arm of silver, a head of gold, 100 wives, 1000 million boys and girls, his urine is Rhine wine, and his feces are gold."

These illustrations might be repeated *ad infinitum*. They are to be found in richer or poorer elaboration, in shorter or longer intervals of excitement, in this exalted euphoric type, but one may see a hundred paretics, as a general practitioner may see them, in the early stages, and never get a ghost of an idea of such experiences. Of this 100, some time, sooner or later, 25 to 30 of them will be liable to exhibit the exalted, euphoric, megalomaniac picture here indicated rather than described.

Consciousness is usually much clouded in this type, especially while the delusional projections are in their full growth. Time, place, the great world, is a dreamy, far-off world of little moment to the mind engaged in its ambitious program. Continuity of thought is practically impossible, and chaos and anarchy exist. In such minds, hallucinations are frequent.

The mood is happy, overflowing with good deeds and generosity, and all embracing in its brotherly love. But coherence is not to be expected. Hypochondriacal ideas—worms in the head—may rest in bizarre connection with the patient being a great philosopher, a Shakespeare, etc., and changes in mood are of frequent occurrence. Weeping follows ecstasy, and is replaced by beatific, sublime happiness.

Sudden, passionate excitement leaps up under restraint, to subside, or to be diverted by such a trifle as a falling leaf, or a ring at the door bell.

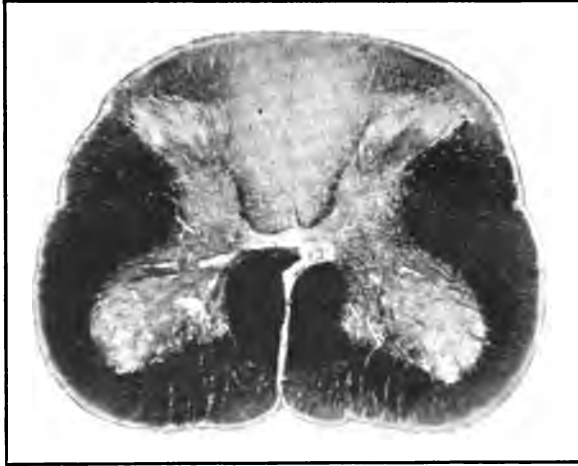
The great psychomotor excitement is a striking feature, and one difficult to manage. These patients walk miles, are on the go, meeting people, busily engaged in everybody's business, making plans for self and others, and, when confined, the limits of a parietic's violence knows no bounds. He is transformed into a raving animal.

Throughout all of the excitement, divertibility, constant changing of plans, mixture of silly pleasure and superficial sadness, there is the note of great deterioration in the intellectual sphere, which shows particularly in the conventions relative to one's person. Carelessness in dress, uncleanness, grossness in eating, loss of finer susceptibilities, coarse expressions, frank immoralities—these are but a few of the possibilities in such lax conduct. .

In watching such patients from day to day, one is struck by the immense variability in the picture. The ideas of grandeur may all vanish, the patient denies he ever said any such thing, he may get angry in a dispute over the matter, and then launch into a magnificent, grandiloquent invective of colossal outlines.

PLATE XXVII

Fig. 1



Paresis with Tabetic Changes in Spinal Cord.

Fig. 2



Brain of Juvenile Paretic Showing Marked Atrophy.

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As the dementia increases, these large ideas may entirely disappear, or be preserved, and appear on the surface only as a few words, or murmurs, "good to eat," "fine women," "millions," etc.

Finally, in the later stages, the patients all sink to a more or less common level—"sans everything."

Among the expansive forms may be found the quick, galloping patients who die within a short time. Increasing experience seems to show, however, that these excited types indicate a very severe reactive process, and hence, if they do not die in the height of the reaction (galloping cases), they provide the greater number of the more stationary and protracted forms—those who make a partial recovery with defect, and who later disintegrate. Remissions seem to be common in this type as well.

4. **Agitated Forms.**—Those patients who show a predominant motor activity in the beginning may be said to be grouped here. Great restlessness runs through the entire picture. The mental content is very variable—euphoric, depressed, hypochondriacal, mood colorations flit in and out. Galloping cases are usually grouped here, where an extremely rapid and fatal course is present.

It is a subgroup of the preceding type, only artificially separated off by reason of the more consistently persistent psychomotor restlessness. Remissions are frequent, as are also the apoplectiform and epileptiform attacks. The pathological process simply has a wider extension in the motor areas.

The acute delirious cases, somewhat resembling delirium tremens of alcoholism, and independent of it, are arranged by Kraepelin in the agitated group.

5. **Irregular Types.**—Lissauer, etc. Those patients showing irregular forms of development; neurosymptomatic groupings, hemiplegias, etc., are here brought together. The hemiplegic and taboparetic groups are the more frequent.

Taboparesis.—Taboparesis is the more striking of these irregular forms and deserves a further outlining.

It will be shown later in the section on pathology that in paresis changes are always to be found in the spinal cord. These may be general, but for the most part show the classical alterations as seen in tabes. A small number of these patients first show signs of tabes; it may be at least ten to twenty years before the development of the cerebral localization of the disease which is symbolized under the term paresis. In the majority of the patients who show the combination, the two localizations, or rather the extension of the disorder to the whole cerebrospinal axis, occurs more or less simultaneously.

It has been assumed by many, especially by neurologists (Schaffer, for summary, 1912) that tabes may be regarded as a spinal paresis and paresis a cerebral tabes; that is, the disease varies only by reason of the greater severity of the process in the one or the other localization.

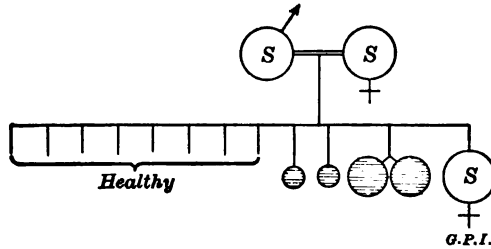
Kraepelin, on the other hand, accentuates the opposing psychiatric view, that whereas, the two disorders are undoubtedly fundamentally

syphilitic, yet they are two different kinds of processes, and that when the symptoms of tabes are added to paresis the changes in the cord are not exactly similar to those found in tabes limited to the cord. The different findings in the cerebrospinal fluid in the two disorders would point to some sort of a difference as well.

The matter still rests on the knees of the gods.

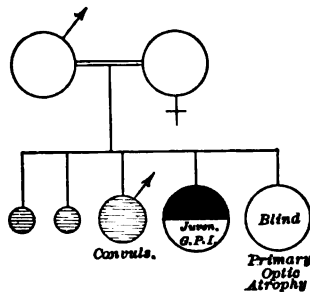
Clinically, taboparesis shows in a combination of the symptoms observed in the two forms. Raymond and Nageotte would have it that every paretic would show tabetic signs if he lived long enough. In those patients with pronounced tabetic onset one finds the frequent pupillary anomalies, the diminution or loss of the patellar reflexes, Romberg sign, ataxia of lower or upper extremities, or both, hypotonia, lancinating pains, crises, and arthropathies. These are found in the more definite taboparetics closely associated with the psychical disturbances already outlined. In the more classical tabetic patients the mental disturbances, to which Cassirer and O. Meyer have devoted their attention, are very distinct from those of paresis.

FIG. 20



Family tree showing influence of syphilis in parents with abortions, dead children, and infantile paresis. Infection took place after birth of eighth healthy child. (Mott.)

FIG. 21



Both parents syphilitic; two miscarriage; child dead of convulsions at fifteen months; juvenile paresis at age of fourteen; last child blind. (Mott.)

6. Juvenile Paresis.—This form is quite distinct. It was apparently first recognized as late as 1877 by Clouston. It appears at the present

time not infrequently, since the Wassermann-Plaut findings offer such certain criteria for its determination. Such tests seem necessary, since the clinical picture may be so extremely variable—hence it was overlooked—many patients died as “imbeciles.”

Here the patient may make a comparatively normal development to five years of age—certain non-developmental forms probably belong here, but are now disregarded. Then the child's mentality seems to drop. In older children, ten to sixteen, this drop is more apparent. Immature, poor memory, bad motor development and gradual dementia appear. Childishness, fabrication, excitements and depressions, fears and anxieties, are frequent. Epileptiform convulsions appear—many juvenile paretics are gathered into the almshouses and epileptic colonies as “epileptics with feeble-mindedness”—and after a course of three or four years, with gradually deepening mental disintegration, the patient dies. The histopathological changes are identical with those of the adult form.

Course.—The average course of the disease, so far as its more active features are concerned, varies from two to three years. Patients may die in the first convulsion previous to which no disease has been suspected; this is extremely exceptional; a few galloping cases die within six months; on the other hand there are certain very rare instances of a very long course. Nissl examined the cortex of a patient who died twenty-eight years after the onset of symptoms. It had all of the signs of a true paresis. Alzheimer has reported a case of thirty-two years' duration, and the literature contains numerous references to cases in which early signs of syphilis of the nervous system have been present ten, fifteen, twenty years before final death by paresis. Notwithstanding these anomalies, it is fairly certain that 50 per cent. of paretics die within two years of the frank onset of the disease.

Attention has already been directed to the fact that it is generally considered that paresis runs a uniformly fatal course. While what seems to be evidence that this is not absolutely true is accumulating, still the most sanguine optimist approaches the question of the prognosis of paresis with some hesitancy.

The study of the cerebrospinal fluid has shown that the cell changes, so prominent in adult paresis, may exist for years before the marked clinical manifestations. Certain syphilitic accidents, especially in the cranial nerve innervations, diplopia, ptosis, Argyll-Robertson, dizziness, fainting attacks, passing epileptiform seizures, are known to occur four to ten years preceding a paresis. They may also occur without any later developing paresis.

Even the mental signs, so prominent in paresis, may show themselves very insidiously, and take several years to develop into a fully marked parietic picture. The question of a syphilitic neurasthenia, as a precursor of paresis, is one which is by no means settled. Again it should be borne in mind that just as cranial nerve precursors are known, so also may psychical precursors be present in the form of attacks of acute

excitement—a manic attack or an acute depression—such may be a true syphilitic accident, and antecedent to, or preparatory of a paresis. Experience seems to show that such mental events antecedent to paresis are rarer than the neurological events, but it may be solely because attention has not yet been directed to them.

Remissions constitute a very striking picture in paresis, and, so far as figures can show, such indicate that they occur in about 20 per cent. of all cases, although long remissions, very distinct remission, in the sense of marked mental amelioration, occur much less often. Gaupp places a very low percentage on long remissions—*i. e.*, one or two years—namely, 10 per cent.

In the remission some patients obtain much insight into their former state, and often wonder how they could ever have been so nonsensical.

In the majority of cases, the recovery is only partial. Careful intelligence tests will reveal marked defect, yet to the layman, or even to the physician, the patient seems perfectly well. These remissions always constitute a danger point in this disease, as previous restrictive measures are not infrequently let up, and the patient, in his new-found freedom, and still not normal state, is very apt to commit some foolishness.

These remissions last a variable length of time, from a few months to several years, and in this period certain patients are known to do valuable work, even in the more cultivated walks of life, painting, sculpture, etc. Those remissions which last a few years, however, are the exceptions, though remissions of over ten or even fifteen years do occur in undoubted paresis; or, shall it be said—periods of health between a syphilitic antecedent, and a parasymphilitic consequent; in a sense analogous to diphtheria—diphtheritic palsy. For, it must be borne in mind that, in all of those cases under discussion, those of Gaupp, Alzheimer, Nasse, Kraepelin, Tuczek, Halban, and others no cerebrospinal fluid studies were made in the earlier period of the attack, and, as it has been pointed out, both neurological and psychotic antecedents of syphilitic origin may precede paresis, or occur independently of it, and remain as cured, isolated, syphilitic phenomena.

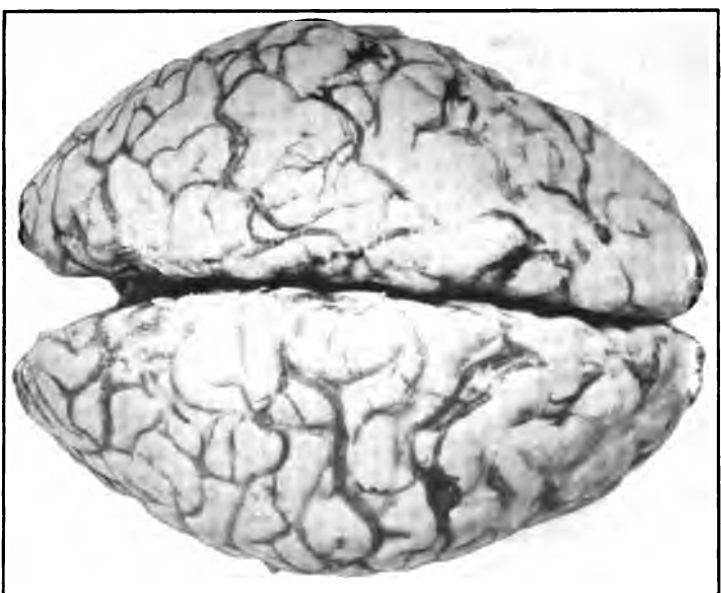
At all events, laying aside this lack of technical completeness in the picture, it seems highly probable that certain rare instances have occurred, where paretics have recovered, or have run such a protracted course as to be logically regarded as having made a recovery. The cytological and serological gaps are now being filled in, and are beginning to be available in sharpening our criteria on this question (Plaut).

Kraepelin and Gaupp have shown that in the majority of cases the long protracted cases, and the cured cases, so-called, are cerebrospinal syphilis.

Nearly all of the patients with paresis die within five years after the decided outbreak of the disease. Kraepelin's series of 244 cases showed the following death rate: I, 51; II, 63; III, 52; IV, 4; V, 22; VI, 4; VII, 5; VIII, 2; IX, 2; X, 1; XIV, 1.

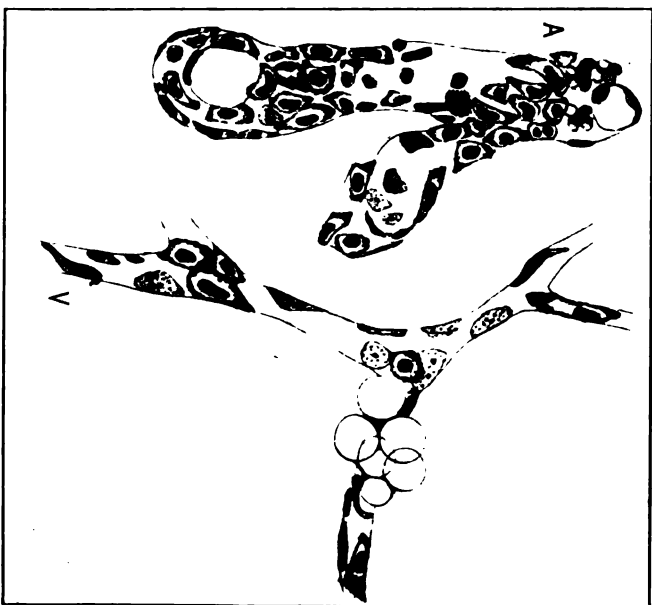
PLATE XXVIII

Fig. 1



Brain of a Paralytic with Opacity, Thickening
of Pia. (Lafora.)

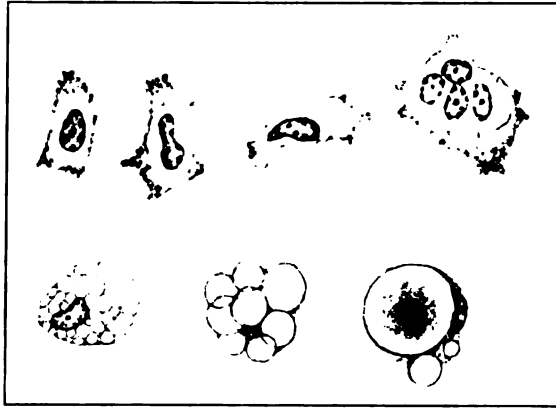
Fig. 2



Plasma Cells about Bloodvessels. (Lafora.)

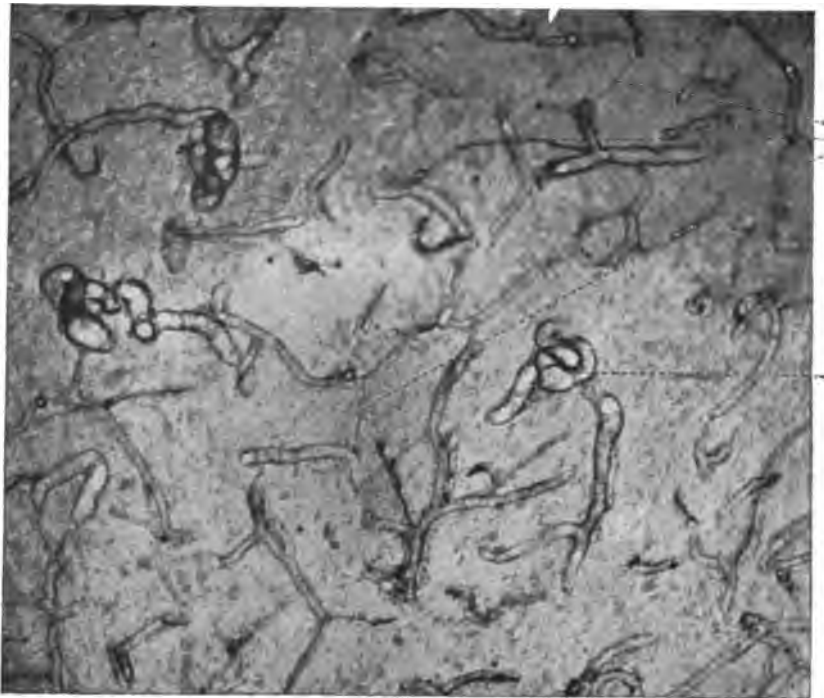
PLATE XXIX

Fig. 1



Paresis. Plasma Cells Degenerating. (Lafora.)

Fig. 2



Paresis. New Vessel Formation. (Kraepelin.)

A large number of statistics show the average duration at twenty-four to twenty-eight months. The short periods also show great variability. Certain patients are known to die in their first apopleciform seizure—paresis never having been suspected—others run an acute course of a few months.

Death may occur in a seizure, from choking, insufflation, pneumonia, intercurrent disease, or from general debility and secondary infection.

Histopathology.—General paresis is a widespread reaction to the syphilitic virus. That this reactive, productive result may be due to other factors than syphilis alone is a large question which cannot be entered upon here. The work of Nissl and Alzheimer has seemed to show that certain anatomical pictures are present in paresis which are not found in other types of syphilitic disease. This dictum, while apparently still not overthrown, is actively disputed, and in certain regards seems to be in need of modification. More instances of the simultaneous occurrence of tertiary syphilitic and paretic changes within the brain are accumulating, and it seems not unlikely that certain modifications must be made in the formula that regards paresis as a disease process absolutely separable from tertiary cerebral syphilis (Straussler). That this point of view has definite therapeutic relations is obvious.

The disease process in paresis is not limited to the nervous structures alone. It is present throughout the entire body, but to regard it as a disease of the bodily organs, independent of nervous structures, does not seem to be justified. It has never come to our notice that a diagnosis of paresis has been made without nervous cell changes. The statement then that paresis is a physical disorder, with mental accompaniments and not a mental disorder, is only a play on words.

The old classical teaching regarding the gross lesions in the bones and dura may be swept aside. They are true for other things than paresis and may not be found in true paresis. The pial infiltration is more characteristic. Periarteritis and lymphocytic infiltration is the rule.

Plasma cells are frequent in and around the small vessels. Plasma cells, as plasma cells, are alone not diagnostic. Within the cortex, many changes are taking place. They with the pial changes must be regarded in their totality. New bloodvessel formation is frequent in both the pia and the upper layers of the cortex, and regressive changes accompany the productive reactive growth.

In the brains of paretics, who have been sick for some time, there is marked atrophy of the cortex, the reduction being particularly noticeable in the frontal lobes (80 per cent. Alzheimer). Other gyri or lobes are involved, usually in minor degree. General diffuse atrophy is present in the severer, long-standing cases. The ventricles are usually dilated, the ependyma being swollen and rich in neuroglia undergoing hyaline degenerations.

Along with this active proliferation of new bloodvessels, which takes place in the cortex very irregularly and in much variation as to richness

and location, the elastic tissue network about the vessels shows striking alterations. The adventitial cells increase, there is a dilatation and infiltration of the adventitial lymph spaces. Lymphocytes, plasma cells, mast cells occur in the infiltrate. Rod cells of Nissl are a frequent finding in these areas of new vessel proliferation, with regressive vascular degeneration.

The ganglion cell changes are widespread and very variable. Total destruction with neuroglial replacement is present in certain areas; others show less ganglion cell destruction. The cell architecture is markedly impoverished.

The ganglion cell changes vary from those of acute swellings to the Nissl grave alterations which point to total destruction. These cortical changes are more frequent in the frontal regions, but great variability is the rule.

The axis-cylinders undergo degeneration in like proportion to that of the cells, those of the third and second cellular layers bearing the brunt of the degenerative process. The neurofibrils show pathological waviness, beading, crumbling, and granular dissolution, particularly accentuated in the peripheral networks about the nucleus, and finally in the cells; with grave Nissl disease the fibrils have entirely vanished.

Neuroglia replacement as well as new glia formation is common and widespread. The external layers of the cerebral and cerebellar cortex are particularly infiltrated. A similar grouping of neuroglia cells is found about the bloodvessels. Degenerative alterations are present in the neuroglia as well.

Similar changes are found in the basal ganglia, the cerebellum, pons, medulla, and spinal cord, but their description lies outside of the scope of this work.

Similarly it can be mentioned only that changes of a definite character are found in the sympathetic ganglia, peripheral nerves, and other organs of the body. Thus the aorta is involved in at least 75 to 80 per cent. of all paretics, and periarteritic changes are noted within the organs throughout the entire body.

Treatment of Paresis.—The treatment of paresis is as yet an uncertain chapter in psychiatry. An attempt will be made here to discuss the treatment from several points of view. I shall discuss:

1. The possible abortion of paresis.
2. Treatment of early stages as a syphilitic disease; other therapeutic suggestions, as secondary infection, etc.
3. The medico-legal situation and its needs.
4. The treatment of the disordered conduct—particularly excitement, attacks, and general management.

1. **Possible Abortion of Paresis.**—In a following section on tabes, this subject is considered *in extenso*. The position is there taken that a probable paresis may be diagnosticated months, if not years, before the actual onset of the disease, by means of the examination of the cerebrospinal fluid and of the blood. The four reactions are positive. The fluid being positive in small quantities, 0.05 to 0.2 c.c.

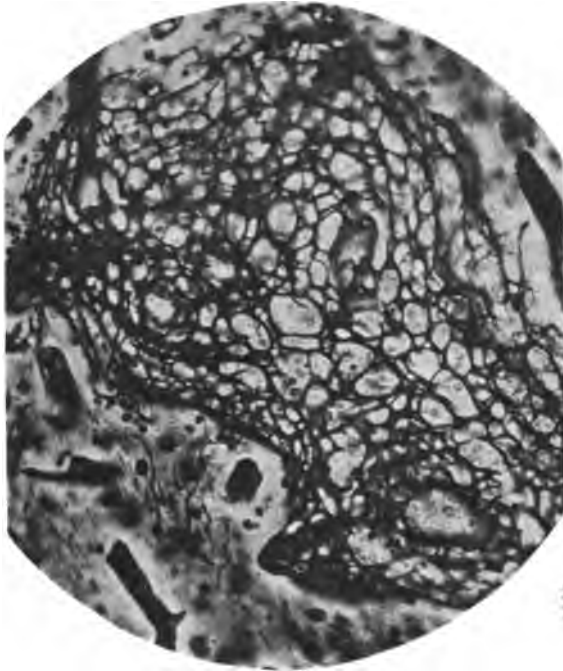
PLATE XXX

Fig. 1



Brain of a Paretic Showing Marked Atrophy. Peeling of the Cortex due to Adhesions of the Pia.

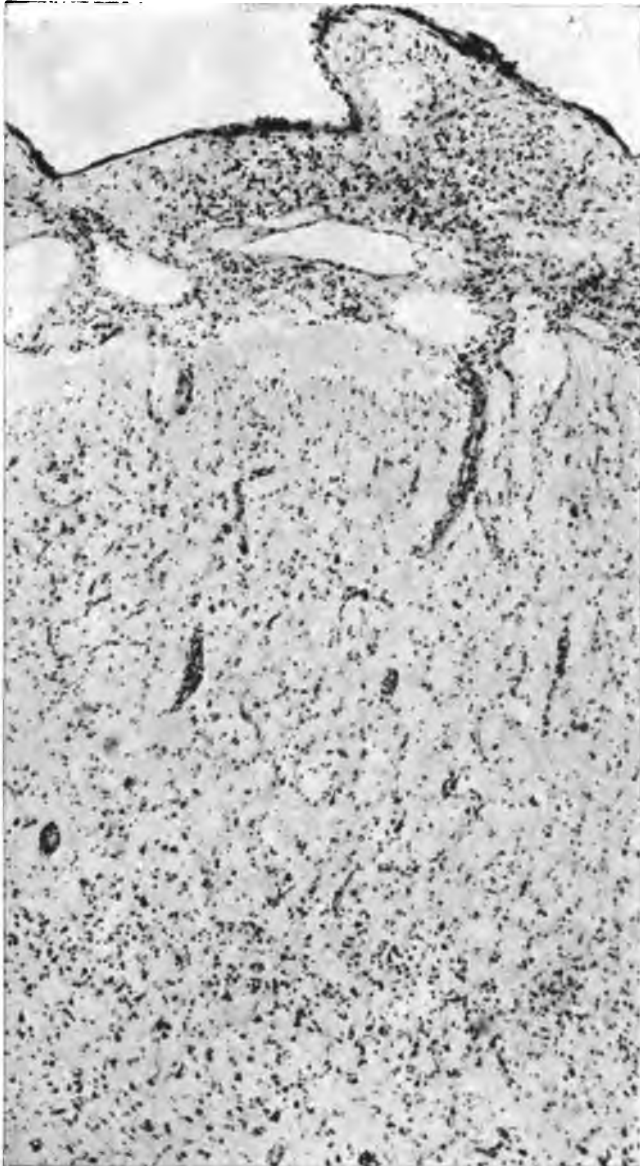
Fig. 2



Organization of Virchow-Robin Lymph Spaces in General Paresis. Tannin Silver. (Achucarro.)

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PLATE XXXI



Devastation of Cortex in Paresis. (Kraepelin.)

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The positive occurrence of the four reactions may also be encountered, however, in active fulminating states of cerebrospinal syphilis, particularly of those forms with meningeal inflammatory changes. This introduces a doubt of diagnosis for a small number of cases. It is, however, a positive indication for an active antisyphilitic treatment, and hence runs parallel with the goal to be gained. It is the author's personal conviction that an energetic antisyphilitic treatment as outlined in the section on Tabes (see page 369) in the biological stage may abort a general paresis. Logically it may be claimed that one begs the question. How can a paresis be aborted when there is no uncontrovertible evidence that a paresis and not a cerebrospinal syphilis will develop?

Historically it will be recalled that Fournier laid strong emphasis upon his opinion that by systematic and thorough treatment of syphilis, paresis might be prevented. He attributes his previously mentioned experience regarding the fatal course of the apparently light cases of syphilis to the circumstance that such cases seldom or never receive sufficient treatment. According to his reckoning only 5 per cent. of paretics have gone through a course of treatment sufficient for all requirements. He, therefore, advises in the first two years after infection a thorough course of mercury which should be repeated in the fifth, seventh, and eighth years, and even longer.

Most other writers upon the preventive action of mercurial treatment are much less convinced. Kiss found that 77.1 per cent. of his paretics had had antisyphilitic treatment; Schüster, of whose patients 17 to 19 per cent. had had several courses of treatment, thinks that no influence either upon the frequency of paresis or upon the complement-fixation can be demonstrated. Of the Dalldorf patients (Junius and Arndt) 45.2 per cent. had had antisyphilitic treatment, often only locally. Marcus found among his paretics that the treatment had been but partly carried out, and Kraepelin writes that in the great majority of his cases only a single course of inunctions or injections, often enough of only one dose, were given. Hudovering and Gutzmann determined that among 28 cases of metasymphilitic and late syphilitic brain and cord affections, a half had received no treatment, and only 5 a sufficient amount. It will require new and extended experience to settle definitely the question of the preventive value of an early and thorough treatment of syphilis. It has been shown that active antisyphilitic treatment cannot influence a paresis unfavorably, hence, writes Kraepelin, if the biological tests point to a continuance of the action of the syphilitic virus, such an antisyphilitic treatment should be carried out.

Juvenile Paresis.—The same position is taken for the abortion of a *juvenile paresis*, and it is justifiable to demand a Wassermann blood test for all children of parents who were syphilitic before the birth of these children.

This is particularly true for the children of paretics or tabetics, or those with nervous syphilis generally.

This phase of the situation is discussed at length in the section on Congenital Syphilis (see p. 415).

Given a positive blood Wassermann, a cerebrospinal fluid examination should be made, and active antisyphilitic treatment carried out. To continue until a negative Wassermann is a settled result is too ideal a program.

Children react well to inunctions. These may be combined with neosalvarsan. The serum reaction should be utilized to answer the question, How much to give? This criterion should not be carried, however, to foolish lengths, especially in view of the many uncertainties relative to the Wassermann reaction.

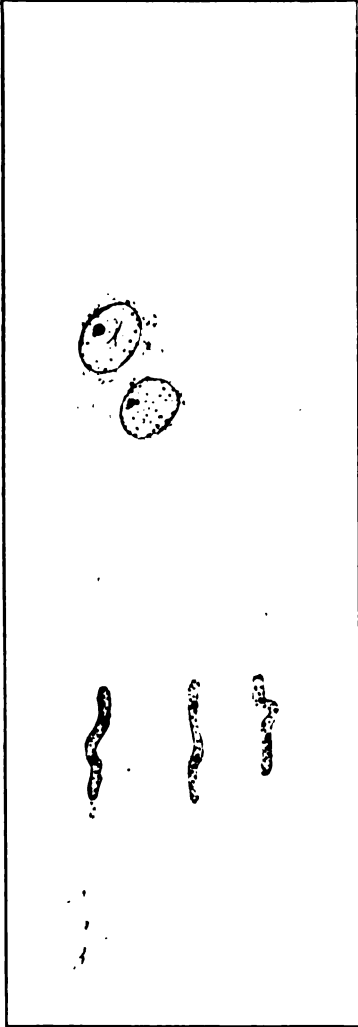
Should these suspects of a potential juvenile paresis be given salvarsan or its congeners? The author's belief is that there are no reliable data as yet to support any positive conclusions. The general situation must guide one. In the presence of evidence of active syphilis, our belief is that salvarsan is more valuable than mercury, and its use not more dangerous. The potential juvenile paretic should then receive a thorough combined antisyphilitic cure, given in a manner best calculated to avoid pain. A combined inunction and salvarsan therapy can thus be carried out, as injections are usually fought against so unreasonably by young children.

2. Treatment of Paresis as a Syphilitic Disease.—The treatment of paresis as a syphilitic disease, *i. e.*, for the syphilis, presents greater difficulties in its early, and by this is meant "recognizable" stages. The unusually grave prognosis of paresis, with its rapid course, affords a sharper criterion of the value of treatment than does tabes. On the other hand, diagnostic difficulties are more in evidence in paresis. Cerebrospinal syphilis, brain tumor, cerebral arteriosclerosis, complicated alcohol pictures, syphilis in dementia præcox, these present situations which are separable from paresis with extreme difficulty. Fortunately, the serobiological tests have offered additional criteria by which closer approximations can be made. These have been discussed. Up to the present time, however, thorough treatment with accurate control through the serum and cerebrospinal fluid reactions has been carried out for too short a time to enable one to state what the outcome is going to be as far as a paresis is concerned.

The present-day reports, under the revised conditions, from various sources which tend to show the results are extremely variable. In the main it may be stated that recent experiences show that the results of extensive treatment by the newer remedies, salvarsan, neosalvarsan, alone or combined with mercury, are suggestive of possible good to be arrived at in certain cases. In general, however, the general results have not been as apparently promising as in tabes. It is highly desirable that all who have reported their cases should report the outcome. Thus far only two or three years have elapsed since completely observed paretic patients have been under treatment by the newer arsenical preparations. Certain newer methods, especially those of Swift and Ellis, seem to even offer more hope than any heretofore.

PLATE XXXII

Fig. 1



Ganglion Cell Degeneration and Rod Cells in Paresis. (Lafora.)

Fig. 2



Axis Cylinder Degeneration in Paresis. (Kraepelin.)

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A patient upon whom a diagnosis of paresis has been made, upon the evidence already outlined in preceding sections, is entitled to a vigorous antisyphilitic treatment. There are certain situations, however, which should be taken into consideration which are here categorically outlined: (a) The patient may never have received any antisyphilitic treatment; (b) antisyphilitic treatment may have been merely perfunctory or manifestly insufficient; (c) antisyphilitic treatment may have been interfered with on account of idiosyncrasies to mercury or to iodides; (d) the diagnosis may be incorrect—particularly to be considered are brain tumors including gummata; cerebral syphilis, arterial or other type; arteriosclerosis, non-syphilitic; (e) the patient may be in an advanced state with signs of widespread destruction; (f) the patient may be in a state of remission.

Before advising or beginning treatment for a patient with paresis these preceding conditions must be cleared up. The practitioner in treating a patient with paresis has a double duty to perform; he has the practical duty to his patient, and that to his fellow workers relative to the results obtained under fairly accurate conditions. In a field as yet so dark as that of syphilis of the nervous system in general the accumulation of accurate data is of the greatest importance, both from the standpoint of practice and for theoretical consideration.

Form of Treatment.—What type of therapy seems advisable under the several conditions outlined. I shall not discuss the merits of the different types; these have been entered into exhaustively in preceding sections.

The general trend of present-day opinion is that a combined mercurial and salvarsan therapy gives better results in paresis than does mercury or salvarsan alone. Just what is meant by "better results" will be summarized later.

Then to such patients—under paragraph (a), *i. e.*, those with beginning paresis—not under section (e) with widespread signs of degenerative nerve tissue—one should advise or carry out an active combined treatment. The details of such an active combined therapy have been given under the section on Cerebral Syphilis. These patients—under condition (b)—are entitled to the same advice and the same therapy. For those under (c) treatment by salvarsan alone is advisable. For all the intraspinal treatment of Swift and Ellis is advisable.

For those in whom the diagnosis is particularly difficult (d) it may be said that at no time has it been shown that a combined mercury and arsenic therapy is more dangerous than the conditions for which one is being treated. It is true deaths in paresis were reported in the early days as having been due to salvarsan. A few did die, there is no doubt of this—but there is much doubt as to whether the salvarsan had anything to do with it. In fact, practically all of the recent evidence tends to show that other conditions were responsible for the results, rather than the salvarsan. (Nonne, *loc. cit.*; Benario, *loc. cit.*) The Herxheimer reaction, neurorecidive, are possible in paresis, and have

been observed. These point above all to the syphilis present, and afford the necessary evidence to warrant further vigorous attack. If a course of combined mercury and salvarsan will tend to clear up the localization of a cerebral tumor (gumma) through increase of localizing signs, so much the gain for the patient, for then an adequate surgical operation can remove that which no amount of syphilitic remedies can break down or destroy.

If it becomes evident that the lesion is purely an arteriosclerotic one from other causes (heredity, alcohol, or what not) the combined treatment has done no harm—save an economic one possibly. This feature should have been discussed by the physician previously and the responsibility placed before the patient—or in the event of his mental incapacity to those most entitled to consider it. This is a duty one owes to one's conscience.

Should one advise or embark on a course of active antisymphilitic therapy for an advanced parietic? What is an advanced parietic? This must first be answered. The clinical course of paresis is extremely unorthodox. A patient in apparent health may suddenly develop convulsions, be at death's door for a month or so, will appear like a most "advanced" stage of paresis, may have all of the laboratory signs of paresis; may die, and at autopsy one finds a cerebral syphilis, with meningeal involvement. Evidently these are not "advanced" parietics; they might have been saved by prompt treatment. By advanced parietics is here meant those bedridden patients in whom the disintegration has been going on for years, *i. e.*, three to four years, in whom deterioration has been steadily advancing and has reached a profound stage; especially in some who have had a remission, and the advance has been resumed.

Should such patients be treated in the intense manner? Experience seems to show that it is fruitless to do anything with this class of patients. Such a decision should be reached, however, only after painstaking analysis. Many surprises have been the lot of those who have seen some of these hopeless, advanced parietics come to autopsy.

Treatment in Remissions.—As to the treatment in the remissions, *i. e.*, provided the remission has not occurred following the treatment—are these to be interpreted as natural attempts at cure? In some instances this seems to be the case, since in many the spinal fluid lymphocytes diminish, although the Wassermann reaction remains more or less constant. Although a remission may be regarded in the light of a spontaneous attempt at repair, and hence a well-adapted time to attack the disease, the present available data are much too fragmentary to permit conclusions.

The special danger to which attention should be called, however, is not to regard the remission as a signal for leaving well enough alone. If an antisymphilitic course has been decided upon, the occurrence of a remission should not in any way interfere with the carrying on of the treatment. It is a blind and stupid optimism that would regard such a patient as having recovered; to permit him to resume his civil

rights without restriction; to send him abroad as a neurasthenic, or to allow many of the foolish things that friends suggest in this at times very perplexing situation.

The categorical list here suggested has been exhausted, but it does not cover all of the possible situations that may arise in the consideration of the paretic—so far as antisyphilitic treatment is concerned. Attention is called to this, for at any moment a situation may arise in this actively progressing field that will require a shift of viewpoint in all of the points brought up.

Summary of Salvarsan Therapy.—A word then as to one of these newer situations which has done much to provoke research in this almost hopeless field—namely, salvarsan therapy. What is the best summary of opinion that can be offered at the present time? Collective judgment is necessary, since an individual is so small a unit in our large social organism.

Nonne's summary of the intravenous salvarsan therapy is here given. I quote him more or less verbatim (p. 227): "Finally, I come to the consideration of the salvarsan treatment of paresis. Here hopes were not slight. Practically all observers said or wrote that, although on *a priori* lines, there was little to be expected in this disease, yet—and the opinion was general—one could at least hope that the disease could be brought to a standstill, that its character might be bettered, that the remission might come on sooner or last longer, and the fact is that nearly all investigators who treated the true syphilitic diseases of the nervous system and tabes with salvarsan also took on paresis. Ehrlich's warning that paretics in advanced degenerative stages were ruled out of consideration was not followed, for a search of the literature shows that cases of paresis (as also of tabes) in all stages were subjected to salvarsan therapy in all parts of the world.

"It can now be established that salvarsan therapy has done no harm even to the patients in advanced stages of paresis, if one would exclude the single case of Jorgensen, in which an acute arsenical poisoning took place in a very advanced paretic, and which on section showed no disease of the internal organs. It should be specially noted that previously in paresis conditions which must be interpreted as neuro-recursive have not been known. Cases have been noted, as for instance by Treupel, in which conditions of severe excitement, which necessitated restraint have followed the treatment by means of salvarsan. I have had two such cases in my own experience, but such are conditions which are known to be possible in any case of paresis with or without treatment. It should be said, however, that the publications are very recent, and that the time for observation is still short for all cases, and that certain reports are excellent examples of what they should not be. Thus one author writes: 'A woman in whom a beginning paresis was diagnosed, *i. e.*, feared, was completely cured in ten days.'

"The reflection of the numerous and widespread communications is that salvarsan is no more a curative for paresis than is mercury or iodide. Treatment by salvarsan has shown remissions, which came

on quickly and were very marked, showing marked raising of the psychical state, improvement in speech and writing and of the physical signs. No cases are known, however, which are more convincing than those which experienced practitioners have already experienced in paresis. Our conclusion, so far as paresis is concerned, is as follows: In incipient cases of paresis the treatment by salvarsan is permissible. In those patients for whom mercury has been of service, and by whom mercury is no longer tolerated, salvarsan is indicated. In patients with advanced paresis, who have either not been treated or for whom mercury has been of no service, treatment by salvarsan is useless."

The situation for intraspinal salvarsan therapy so far as paresis is concerned is yet too recent to permit of dogmatic presentation. The number of parietic patients as yet treated by Swift and Ellis is too few to permit more than a showing of the results in one patient. These point to very hopeful indications. The most marked improvement here is the reduction of cells from 98 to 4, and the need for larger quantities of fluid to show a positive Wassermann reaction. Clinically, there is improvement. (See History of M. N., page 365.)

The sorry position in paresis is partly explained by the histological findings. Reference to Plate XXX, Fig. 2, shows the infiltrated Robin-Virchow lymph spaces. In paresis these are filled with an organized new growth which effectually stops off all lymph flow. They also can be assumed to block off all therapeutic stuffs which might flow in the lymph spaces. Furthermore, a reference to the Moore-Noguchi findings of the *Treponema pallidum* shows them to be at a distance from bloodvessels and lymph spaces. They have become inaccessible to all therapeutic agents. Paresis then is eminently the problem of the inaccessible spirochete. Whether intraspinal, perhaps intracerebral, therapy will reach the organism is the problem of the immediate future. Introduction of sera similar to those of Swift and Ellis directly into the cerebral vesicles may offer some results in spite of technical difficulties.

The whole question narrows down to this. The *Treponema pallidum* is there. It is safely ensconced as far as possible from all avenues of approach. How can it be gotten at? All other questions such as those of increasing leukocytosis, nuclein therapy, accessory bacterial infections, etc., become insignificant in view of the finding of the organism.

Other Forms of Treatment.—Other treatments for paresis have been suggested. Among them may be mentioned those that would set up an acute reaction to some septic organism. Wagner von Jauregg was among the earlier experimenters in this line. He combined mercury and iodide with Koch's old tuberculin. Pilcz, carrying out his suggestions, reported some excellent results in 1910 by this method. Remissions were marked in his opinion, and some 26 per cent. of the patients showed more than usually good improvement.

Small doses of tuberculin, gram 0.005 to 0.01, are used in the beginning, the dose being repeated every second day, allowing forty-eight hours for the cessation of the temperature reaction. Increase in

History No. 883. M. N., aged forty-five years; general paresis; duration, one year; syphilis, twenty-one years ago.

Date.	Blood.	Cerebrospinal fluid.				Treatment.		Clinical condition.
		Cells.	Noguchi globulin.	Wassermann reaction.		Intravenous.	Intraspinal.	
				Liver antigen.	Heart cholesterolin antigen.			
1912 Oct. 10	++	46	++	0.05 c.c. ++	Border-line case between cerebral syphilis and general paresis.
Oct. 19	++	0.3 gm. neosalvarsan		
Oct. 29, 30	++	98	++	0.05 c.c. ++	0.75 gm. neosalvarsan	30 c.c. 40% serum	
Nov. 5	++	0.9 gm. neosalvarsan		
Nov. 12	++	0.9 gm. neosalvarsan		
Nov. 19, 20	++	4	+	0.05 c.c. ++	0.9 gm. neosalvarsan	30 c.c. 40% serum	
Nov. 27	++	0.9 gm. neosalvarsan		
Dec. 9	++	0.45 gm. neosalvarsan		
Dec. 14	4	+	0.1 c.c. ++			
1913 Feb. 12	++	0.9 gm. neosalvarsan		Improved.

dosage is regulated according to the temperature, the dose being doubled if the temperature does not rise above 100° F. until doses of 0.3 to 0.5 or even 1.0 tuberculin are given. The method is tedious and up to the present time offers little that is definite.

Later this same author, with Halban, utilized the dead products of purulent organisms, streptococci and staphylococci. Remissions occurred late in the treatment in contrast with the rapidly appearing remissions seen in the tuberculin therapy. Experiments are still in progress, but seem to offer little.

Ford Robertson has utilized his *Bacillus paralyticans* along similar lines. They utilize a vaccine. As yet no attack has been made with success along this line, but experiments are still in progress.

The general hypotheses underlying this type of therapy are that (a) an increase in leukocytes may aid the body in its combat with the spirochetes, and (b) the introduction of other pathogenic organisms hinders the growth of the syphilis organisms.

Injections by nucleins, lecithins, spermins, etc., have been tried with the view to induce leukocytosis. Fischer and Donath have utilized nucleinic acid, sodium nucleinate, and related salts. Every three to five days a half gram of the sodium nucleinate is injected. Peritz utilizes lecithin. With it, distinct alterations in the Wassermann reaction have been observed.

Sera have also been employed. All of these remedies are as yet in the experimental stage.

3. Medico-legal Treatment.—A paretic is always potentially a menace to himself, to his neighbors, and to his property. At any given period, *i. e.*, in the early stages, this may not be in evidence, but at any moment such a patient may suddenly alter in his behavior, and, by antisocial or foolish conduct, jeopardize his own life or that of others, or wreck a fortune, or become involved in scandals of various degrees of complexity or unsavoriness.

Sexual aberrancies are very frequent even in the early stages. These may consist of exhibitionism, homosexuality, incest, shameless conduct with prostitutes. A great variety of entanglements, social, legal, economic, ensue, with the result that human parasites, male as well as female, fasten upon such a victim and literally gnaw him to the bone.

Again, such patients become the prey of promoters, speculators, reformers, etc. Even religious organizations have sucked their quota from paretic patients. Foolish business schemes also dissipate fortunes.

Hence, every paretic should be under some sort of surveillance, so that his person and property may not be put in jeopardy by reason of his disease.

Under the best of circumstances, with all the help that can come from the best forces of social culture, it may be a difficult problem to bring about a maximum of protection with a minimum of deprivation of the rights of the individual, but when the situation becomes complicated by inefficient laws, designing and grasping lawyers, ignorant, arrogant, and inefficient judges, the situation is hopeless. Often the

physician has to close his eyes while the human vultures found in all societies feed upon their victims. This is not an overdrawn picture, though fortunately it is not a common one, because so few of the victims have money enough to make it worth while.

A fuller discussion of medico-legal problems is found in Volume I of this work.

4. Treatment of Symptoms.—The causal treatment of paresis is still in the experimental stage. The symptomatic treatment is about all that can be done, and it aims solely to make a patient comfortable, keep him clean, and prolong a well-nigh hopeless struggle.

Rest, relief from business responsibilities, and a regulation of the affairs of life are the general formula. When patients are having attacks, when they are noisy and excited, when they are uncleanly or suicidal, they should be treated in a sanatorium.

It is folly to try to treat a paretic at home and allow him unrestricted liberty, unless there are special indications which will permit it. The senseless procedure of sending paretics to summer bathing resorts, surrounded by excitement and distractions, "to keep him amused or to rest his mind," as is so frequently done, should be forbidden. Such trips fatigue the patient, make him fretful, the baths exhaust him, and the usual result is one of very rapid deterioration, with frequent epileptiform attacks. Not infrequently the parasites at such resorts fix themselves upon such patients and extremely trying complications—blackmail, etc.—ensue. All these procedures for a neurasthenic paretic are nonsense—only exceeded by a trip to Muldoon's or other resorts of strenuous athletics.

Mild hydrotherapy is not contraindicated—in fact is advisable—but it should consist solely of warm packs, rubbings, etc. Showers, douches, forced streams, etc., are disadvantageous.

The diet should be ample and supporting, and alcohol should be excluded. Tea, coffee, and tobacco are interdicted by most physicians. In moderation, and at the proper time, they do no appreciable harm—possibly with the exception of tobacco.

Excited paretics, especially of the anxiety type, are managed with considerable difficulty. Such should be transferred to quiet surroundings. Baths and packs are indicated to try to allay the excitement. But not infrequently they are inefficient. Continuous baths in a properly fitted bathroom are of great service, but cannot be found outside of the larger, better appointed mental hospitals. Veronal, hyoscine, other narcotics may be necessary to quiet such very noisy patients, but chemical restraint should not take the place of proper nursing and attendance. These patients should be carefully watched, else they hurt themselves. Abrasions serve as ports of entry for infections which spread very rapidly, causing furunculosis, erysipelas, and cellulitis.

In marked senseless excitement Kraepelin has tried to cause improvement by systematic (subcutaneous) infusions, repeated twice daily. Each time 750 c.c. of saline solution are introduced. The treatment

was continued for two weeks without bad effect. The patient who seemed to be doomed to a rapid decline, underwent marked and lasting improvement, so that further use of this procedure in such cases may be warranted.

Donath has recently recommended very warmly the systematic employment of infusions in paresis; he uses, besides 0.85 per cent. saline solution, a complex isotonic solution.

For the treatment of paretic seizures, certain authors have advocated packing the head in ice; in severe convulsions enemas of amylene hydrate (6 grain). This drug may be given subcutaneously in 5 to 10 per cent. solution. If a quick effect is necessary, chloroform narcosis to the point of quieting the motor symptoms may be resorted to.

In cardiac failure stimulants are indicated, such as caffeine, camphor, alcohol in small doses, but especially saline infusions and inhalation of oxygen. None of these remedies have any marked effect, save in certain prolonged status attacks.

The emptying of the bowels and bladder requires assistance by enema or catheterization, usually only in the beginning of an attack; later the functions operate by themselves, although too long a delay may permit distention with resulting paralysis of both organs, after which they will require constant artificial means. Unfortunately the treatment of vesical paralysis is often interfered with by stricture. It is useful to follow catheterization by washing out the bladder (boric acid), which may be given at a cool temperature when there is laxness of the sphincter. At other times than during attacks the evacuation of urine and feces also needs constant attention if one would avoid urine dribbling and loss of control of the rectum. The physician should accustom himself to note, by palpation above the symphysis, the fulness of the bladder in his paralyzed or bedridden paretics on each of his visits. A warm bath at the right time will stimulate the emptying of the bladder. Vesical inflammation requires irrigation and urotropin. In one of Kraepelin's patients, who had already been catheterized for two years, independent micturition was restored by four weeks of irrigation (tannin solution) twice daily, nor was it lost during a paretic attack lasting thirteen days. In this same patient no bed-sores occurred up to the day of death, in spite of deep coma and almost complete pulselessness.

Nourishment should be always given by tube during attacks (necessary only in attacks lasting many days); simply pouring food into the mouth is highly dangerous. If one is careful to clean and disinfect the mouth frequently with a moist cloth (potassium chlorate) and to maintain the moisture of the cornea by regular half-hourly moving of the partly opened eyelids (to prevent ulceration), one will often succeed in keeping the patient alive even through convulsive status attacks lasting eight to fourteen days.

Paretics in the last bedridden stage require great care. It is necessary to attend personally to the cleanliness of the patient, and to watch the taking of food, also, on account of the careless chewing, to give only

finely cut, easily masticated food, and to prevent the greedy gulping of a meal, since a fatal suffocation can easily occur. During convulsions, and in very demented patients, the prevention of bed-sores is of the greatest importance. Sometimes the best means to this end is the continuous bath, with the patient lying on a stretched sheet, or possibly on a water-bed. If this is not practicable, it is of most help to maintain strict cleanliness of the threatened part by frequent washing with cold water or solution of bichloride in alcohol, careful removal of wrinkles, bread crumbs, etc., from the bed, the use of water or air pillows, or lying upon cotton-wool or moss, which quickly absorb urine or other moisture; unfortunately these are often torn up by demented patients. Finally a regular changing of the position of the patient by an attendant is necessary, so that the patient (in severe cases every half-hour, day and night) lies alternately on his side, abdomen, back, etc. This method, devised by von Gudden, which also, to a certain extent, obviates hypostatic pneumonia, has for decades made it possible to almost do away with the otherwise inevitable bed-sores of paretics (10 per cent. die of this cause, according to Mendel) and, in any case, the very dangerous forms are prevented. It is much more difficult to cause the bed-sore to heal when, through insufficient care or neglect, even for a few hours, it has once become started. Since the patients, by their restlessness and tearing the dressing, often render the usual surgical treatment very difficult, such cases make the greatest demands upon the patience and attention of physicians and nurses. Since the employment of the continuous bath by day and night, even such cases have lost most of their dread. The patients take very kindly to the treatment, and the sores heal, with occasional surgical help, without any complications.

6. TABES

The early history of the term *tabes dorsalis* is full of surprises. It was originally used by the ancients as a consumption of the spine, and its chief symptom was a "pituitary" discharge from the penis, the spinal canal being believed in those days to be open from the brain to the bladder. Gonorrhea, gleet, prostaticorrhea, etc., were then diagnosed as *tabes dorsalis*. In the middle ages spinal symptoms were added, but the old conception still held. When Columbus took syphilis to Europe the *tabes* of modern days appeared, but there seemed to be no real glimpses of it until the beginning of the nineteenth century. Before this it was all lumped in the paraplegia, myelitis groups.

Horn (1816), Weidenbach (1817), W. Horn (1827), Decher (1838), and finally Steinthal (1847), Romberg (1851), and Duchenne (1852) have been the chief historical workers that have fashioned the present-day concept.

As was true for paresis, so also for *tabes*, the debate as to the syphilitic etiology was protracted and fiery, but the serocytological work of

Wassermann, Plaut, Nonne, and the researches of Alzheimer, Schaffer, Nageotte, and Noguchi's finding of the spirochete have left little room for doubt. Pseudotabetic pictures, due to injury, various toxemias (alcohol, beriberi, pellagra, diabetes, etc.), are met with not infrequently. These should not be confused with syphilitic tabes, although there are often striking clinical resemblances.

The question as to the ultimate reason why one syphilitic individual develops tabes and ninety-nine do not is yet unsolved. Those interested will find much discussion of this in the literature already quoted. Abi-trophy, exhaustion, embryonal defect, and reduced resistance all beg the question while offering much of speculative interest.

The long list of causes, heat, cold, exposure, etc., one or all, without syphilis are unavailing; with syphilis of what logical use are they?

The symptoms come on from ten to twenty years after infection on the average. Extremes are from four to thirty-five years. The usual ages for infection vary from twenty to forty years. Extremes of fifteen to seventy years are recorded. Certain races with high syphilitic percentage are practically immune to tabes; certain syphilitic prostitutes have been known to have tabes follow in their wake in high percentages of their victims.

These facts with others, especially those coming from the biological tests, the comparative studies of Spielmeyer on sleeping sickness, all serve to sharpen interest in the search for causes for variation in the virulence of certain strains of the organism, as well as to call attention to factors of individual resistance. Therapy must consider both of these factors very intimately. The early formula expressing these facts were: Tabes equals the measure of the attack modified by the resistance. Strümpell's more extended formula is:

$$\text{Tabes} = \frac{\text{Syphilis} + \text{accessory aids}}{\text{Resistance, more or less}}$$

That such a formula is very elementary is evident.

Symptomatology.—A complete presentation of the symptoms of tabes cannot be attempted here. Rarely do two patients with tabes present identical developments, yet there is a general trend which allows a certain schematization.

Pains and Crises.—In nearly 60 per cent. neuralgia-like pains are the forerunners. These are rarely recognized as tabetic in the early stages, but are looked upon by the patient as "rheumatism," and often so treated for years. In these early stages, long before the other symptoms develop—as many as ten years it may be—cytological tests will reveal them to be tabetic.

Then develop, in irregular sequence and in varying degrees, ocular palsies, Argyll-Robertson pupil, hypotonia, ataxias of lower and upper extremities, optic atrophy, lost knee-jerks, lost Achilles-jerks, incontinence of urine, and trophic changes in the joints. A so-called classical case will show all of these—yet tabes may be present with only one or

more symptoms, if the biological tests are positive. A brief summary of these symptoms, viewed individually, is of service.

Pains are present, at one time or other, in fully 90 per cent. of the patients. They are an index, as a rule, of the leptomeningitic or radicular inflammatory changes in and about the posterior roots. In 60 per cent. of the patients these neuralgic pains are initial. They have been known to precede the development of other tabetic signs by twenty-two years, yet the average is about four years. They are most frequent in the sciatic region, in the leg and ankles, but any region of the body may be chosen from the upper cranial nerves to the lowest sacral plexuses. When extremely severe and located in a complex organ they constitute the *crises*. These pains or crises may occur in the trigeminal, in the larynx (laryngeal crises with dyspnea), or esophageal crises with choking. An olfactory localization may occasion a persistent sneezing, and a cochlear or labyrinthine origin cause earache, deafness, dizziness, or irregular Ménière syndromes with nausea, vomiting, nystagmus, and dizziness. The heart may show irregularities in force and frequency. Breathing, respiratory crises are known. Gastric pains and crises are classical and were described among the earlier symptoms by Gull in 1856. Intestinal colics with diarrhea; rectal pain with tenesmus; vesical or urethral pain with strangury; renal colic; ovarian or vulvovaginal crises; these all may be encountered.

The pains are characterized by their sharp, transitory nature. The patients feel acute, stabbing, or darting pains, at first lasting a few seconds or minutes, later often lasting for hours or days. These disappear as rapidly as they came, to recur at irregular intervals, usually lasting longer, and recurring more frequently, causing soreness and great prostration as the process advances. In some a few attacks a year are noted, in others much oftener. Indulgence in alcohol, exposure to fatigue, strain, or cold and wet may provoke or coincide with an attack.

The patient is rendered helpless as a rule and gets to dread the pains. Some patients have committed suicide in order to escape them. Others develop morphinism, and abstinence pains then complicate the tabetic pains. In later years there is a tendency for the pains to abate, but there is more uncertainty than certainty in this.

With pains and crises hyperesthesia is frequent. Anesthetic spots or bands, girdle sensations, irregular and anomalous losses of bony sensibility, of position sense, of thermal sensibility develop in a fair proportion of the cases.

Reflex Disturbances.—Reflex disturbances follow when the pathways conveying muscle sense are involved. These show in a diminution or loss of the knee-jerk, or the Achilles-jerk, the latter being more often lost than the former.

When the pathways conveying joint sensibility, muscle sensibility (deep sensibility) are involved there follows a greater or less amount of *ataxia*. This is usually demonstrated in the Romberg sign, or by

the gait which becomes uncertain; the legs are spread more widely apart, and perform exaggerated motions—being lifted higher and coming down with greater violence.

This tabetic ataxia is rendered worse when the eyes are closed, or when the patient goes about in the dark, as then the accessory aids to distance estimation are withdrawn. On the contrary, it is to be noted that many tabetics with optic atrophy show little or no ataxia, and that many ataxias are improved with oncoming blindness. These anomalous situations suggest some valuable points in therapy. To simply explain them as cervical tabes, or as receding root involvement renders one blind to the possibilities of training by other methods than those of Frenkel, heretofore exclusively employed.

Ataxias may show in a number of muscles other than those used in walking or grasping.

Cranial Nerve Involvements.—Cranial nerve involvements are frequent in tabes. Any may be looked for but the most frequent are present in the pupillary reflexes. These pupillary anomalies are present in at least 75 per cent. of the cases. They may be simple inequalities in the pupil, or irregularity in the outlines, but the most frequent is the Argyll-Robertson pupil in one or both pupils—namely, loss of the light reflex, with preservation of that of accommodation. This pupillary anomaly is discussed in the general section on diagnosis of cerebral syphilis and need not be entered into more at length here.

Ocular palsies, particularly unilateral ptosis, and strabismus are frequent; the third, fourth, and sixth nerves being involved alone, in combination, or in irregular medleys. Facial palsy may be present. Bonnier's syndrome, irregular nystagmus, nausea, vomiting, Ménière's syndrome, are expressions of the involvement of the vestibular paths. Vagus, glossopharyngeal, or hypoglossus involvement cause irregular heart action, taste disturbances, or bulbar ataxias or palsies.

Vesical Disturbances.—Vesical disturbances are frequent, especially weakness. Cystitis is a not uncommon complicating factor. Impotence is usual.

Trophic Changes.—Trophic changes in any joint are to be looked for—the knee, ankle, and wrist are those most often found. Arthropathies of the ribs, jaw, or other joints are infrequent. Fragility is a frequent accompaniment, and breaking a bone is not infrequent in tabes. Certain patients have had attention first called to a tabes by such an occurrence.

Muscular atrophies are also to be observed.

Psychic Disturbances.—Psychic disturbances different from those of paresis may occur in tabes. These are discussed in a section on tabetic psychoses (p. 408).

Course and Progress.—Duchenne (1858) first gave the outlines of the traditional course of the disease. It consisted of a prodromal or preataxic, an ataxic, and a paralytic stage. Such a scheme is of purely arbitrary value, even if not an unusual one. Tabes may develop in so many different ways that no law is absolutely valid. Enough may

PLATE XXXIII

Fig. 1



Tabes. Third and Sixth Nerve Palsy.

Fig. 2



Tabes with Bilateral Ptosis. Operated upon to Hold Eyelid Open.

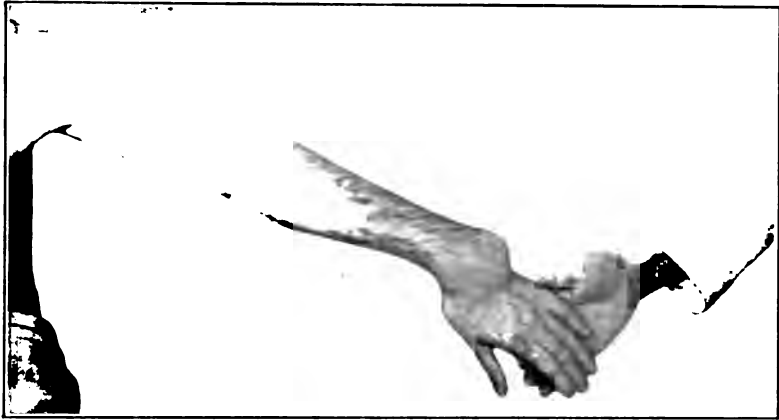
Fig. 3



Tabes. External Rectus Palsy.

PLATE XXXIV

Fig. 1



Tabetic Arthropathy.

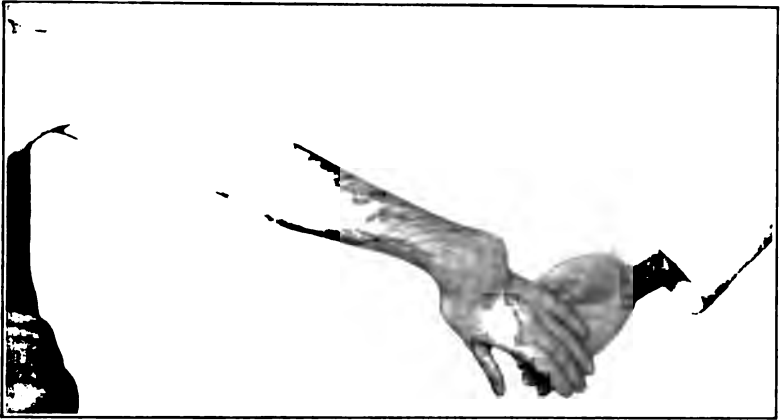
Fig. 2



Tabetic Arthropathy with Destruction of the Joint and Hypotonus.

PLATE XXXIV

Fig. 1



Tabetic Arthropathy.

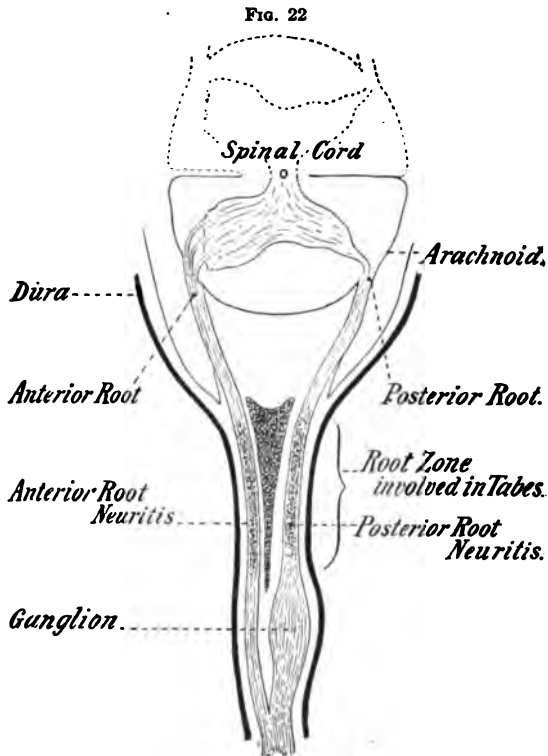
Fig. 2



Tabetic Arthropathy with Destruction of the Joint and Hypotonus.

be said to indicate that a patient may have tabes and yet not have a single so-called neurological sign—his cytobiological reactions show him to be a candidate for tabes. Some patients begin with an acute ataxic course—others with an arthropathy, others with a psychosis. Neurology is not yet in a position to state how valid, “statistically,” Duchenne’s law is. Yet for general purposes it is helpful.

Prognosis.—In general this is sinister, yet not as hopeless as is usually thought. Some patients recover to a certain degree. It cannot be stated that they get absolutely well—especially not if the disease has gone on for years and destructive changes are demonstrable by examination. As will be discussed later, the therapy should begin early, and it is not too much to express the hope that, with means at hand to make much earlier diagnoses, much better results, especially so far as hindering the progress of the process is concerned, may be expected.



Nageotte's scheme representing the chief lesion of tabes in the posterior root zone.

The Treatment of Tabetic Pains and Crises.—Since both the pains and crises are practically identical phenomena, varying quantitatively only, or from the standpoint of localization, they will be considered at the same time.

It has been indicated, in the previous discussion, that the radicular

hypothesis offers the best mode of approach to the problem of the treatment of this series of phenomena.

This general hypothesis may be diagrammatically illustrated by the figure taken from Nageotte. Here one assumes a neuritic process in the posterior root area. Such may arise from the pressure of a meningeal exudate about the roots, or from an endoneural inflammatory or toxic activity, or there may be other unknown or unappreciated factors, and those just referred to may operate alone or in combination. The end result is interpreted as a radiculitis, and the chief accompanying event in consciousness is pain.

It therefore becomes important in all patients suffering from tabetic pains or crises to endeavor accurately to localize the root areas involved. Such an examination entails the following out of a prescribed scheme such as that outlined by the researches of Head and Holmes. The topography of the involved roots should be charted, as they will prove of service in determining whether treatment is accompanied by advances or by regression in the affected areas (Förster, Gulicke, Singer).

It will be recalled that heretofore there has been a marked skepticism regarding the fate of the tabetic and his pains. Such a therapeutic nihilistic standpoint is fortunately being receded from by most present-day observers. Positive results are being obtained, and the uselessness of the nihilistic point of view is being appreciated, even if only as a metapsychological problem.

Importance of Early Treatment.—The newly acquired facts concerning the etiology, the pathological processes, and the localization of these processes, emphasize the importance of early treatment. This naturally can be furthered by an earlier recognition of the character of pains which are of tabetic rather than of so-called rheumatic, gastric, vesical, urethral, renal, autotoxemic or other hazy origins. Enough has been said to emphasize the diagnosis, and especially the aids from cytobiological findings.

This newly gained optimism, however, needs to be tempered with humility, for with each new advance one sees new avenues opened that are in need of investigation. In practical every-day work it would appear that the situation is hopeless, but it is not, and the physician treating a patient with tabes needs a constant store of energy to support his own, and his patient's, at times flickering, hopes.

Should one view the entire tabetic picture as a whole, and should one have in mind absolute cure alone as the only therapeutic goal, it would appear that such hopes must be very meager, but if one directs one's attention to particular symptoms, or is satisfied when an arrest of progress can be brought about, then the situation lightens up considerably. This is particularly true if one is fortunate in obtaining a very early notion of what is going on in the nervous system of a potential tabetic.

This situation is in need of restating, since it is almost the daily experience of the neurologist to find trigeminal tabes treated for months or years as malarial, Gasserian tumor, autotoxemic, etc.; laryngeal

PLATE XXXV

Fig. 1



Tabetic Arthropathy of Ribs.

Fig. 2



Tabetic Changes in Cord in Paresis.

and esophageal tabetic pains as hysteria, and under various neurotic guises; gastric pains and crises as periodic gastritis, cyclical vomiting, hyperchlorhydria, and some score of other designations; visceral crises, rectal and bladder crises under many misappropriate conceptions as well. The numerous fruitless operations which have been performed upon tabetic patients must be interpreted as a part of the suffering necessarily inflicted in the conquest of knowledge—before the cytological findings were known there were no certain or probable guides that could help the profession to avoid such pitfalls—now there is little excuse for them.

Prophylaxis of Tabes.—Inasmuch as the treatment of tabetic pains is for the most part the treatment of tabes in the early stages, a few suggestions may be pertinent relative to the prophylaxis of tabes itself. As to the general prophylaxis of syphilis, some general remarks have already been made.

The formula of Strümpell has already been quoted. Tabes equals syphilis, plus certain accessory factors on the one hand in contest with the greater or less resistance of the patient on the other. In this formula only one fact, *i. e.*, syphilis, is at all definite—even this, when one bears in mind that a microorganism varies greatly as to inherent factors of activity, toxicity, etc., is not as clear as one would like it.

Accessory Factor in Development of Tabes.—*Alcoholism.*—What are the accessory factors? Here one is largely in the field of speculation. Alcoholism at one time was made entirely responsible as the accessory aid. Alcohol has, as is well known, an almost specific action upon the lipid substances of the body, in which substances the nervous tissues are particularly rich.

Most degenerative or catabolic activities of cell life seem to be associated with lipid modification. Expressed in more universally understood terms, one states that alcohol induces a diminished resistance, because it causes neuritic changes, particularly on the sensory neurone side, and the syphilitic plus alcoholic toxic factors determine that the sensory neural elements shall be the special ones to break down.

Such an argument contains truth—hence, syphilitics should be total abstainers. There is abundant evidence from the work of Kraepelin and his students to show that the sensory side of the neural mechanism is extremely sensitive to even the smallest doses of alcohol. Hence, one must be uncompromising in one's attitude on the subject of alcohol in an infected person, at least until further light will show that alcohol has no action upon those neurones which have been demonstrated to bear the brunt in the tabetic process.

Other Toxic Agents.—Alcohol serves as the symbol for a group of toxic agents and any belonging to this group should be excluded as far as possible. Many of the sleep-producing drugs contain the alcohol radical, and it is not unreasonable to limit their use in syphilitics.

Tea, coffee, and tobacco have not been proved as yet to have the same specific action on the sensory neurone, and up to the present time

there is no rational proof that they can be said to provide accessory factors when used in moderation. When used in excess their effects must be made the standard as to one's judgment whether they diminish the resistance factors of the human body or not.

Other special toxic substances must be avoided. Thus a syphilitic exposed to lead, to arsenic, to any poison known to affect the sensory neurones in a manner analogous to alcohol, should be taught to avoid certain definite trades, occupations, etc. Such details should be distinctly laid down and should be founded on precise information—such as may be obtained here in this treatise in the chapters on Alcoholism, Dangerous Trades, etc. One is not privileged to give the patient an indefinite scare, as it is likely to result in inculcating a phobia in the syphilitic mind.

At the same time it must be borne in mind that one can strain at gnats and swallow camels, and tabes and paresis only occur in about 2 per cent. of syphilitics—at the present stage of statistical inquiry; but tabes and paresis, it may be parenthetically remarked, are not the only post-syphilitic nervous results.

At the present time, little definite is known relative to other toxic accessory factors, and one must be on one's guard against admitting hazy, indefinite notions regarding auto-intoxications. Diabetes is one in which the very situation—*i. e.*, sensory neurone poisoning—is an ever present factor, hence, a careful clinical control of the sugar metabolism would logically be reasonable. The advances made in biochemistry within recent years are pertinent in this connection, and may reveal other auto-intoxications as definite as diabetes. At present, however, such other auto-intoxications are nebulous. (See Dakin, Oxidations and Reductions in the Animal Body—Loeb Mechanistic Conception of Life—Die Indicanuria.)

Secondary Infections.—Other possible auxiliary factors are secondary infections. Much speculation has been made in running these to earth, but as yet with no definite success. The most recent of these is the *Bacillus paralyticans* of Ford Robertson. From all that can be gathered at the present time, this bacillus is a universally distributed laboratory saprophyte. The evidence is as yet very inconclusive as to its having anything to do with paresis or tabes.

From what has been said relative to the similarity in types of inflammatory products found in syphilitic meningitis, in cerebrospinal syphilis, in tabes, and in paresis, it hardly seems necessary to invoke any accessory bacterial agent.

As to the factor of resistance in the Strümpell formula, much might be written, but it is all very indefinite. The accessory factors all mentioned might also extend their influence into this field, since toxic agents in general may be said to lower the resistance of the body to other toxic factors—*i. e.*, to the syphilis toxin.

Sexual Excesses.—It has been taught for ages that sexual excesses were causes for tabes and paresis. The origins for this belief are in part made plain by the early meanings attached to the word tabes

dorsalis. Naturally, in a day when a chronic gonorrhea was called tabes dorsalis, sexual factors would be held to play a large role. When the relationship of sexual contacts and syphilitic infection became appreciated, then the same idea—somewhat modified—was reinforced. But logically of course, there is no relation between what properly is meant by sexual excesses and syphilis—ergo not with tabes. In a much later form the idea was transformed as follows: that the drain of much sexual intercourse can produce an actual degeneration of the spinal cord and cause the tabetic lesion. Pathology teaches that this is nonsense. Is there still any truth in the notion that the fatigue of so-called excessive sensuality can reduce the resistance of the cord to the toxin, and thus determine that particular development known as tabes. This also seems foolish in view of the knowledge that the chief lesion is a productive, infiltrative, meningeal inflammation about the roots of the sensory nerves. One can therefore eliminate that widespread teaching of several generations that various forms of modified sexual intercourse—fellatio, bestiality, cunni linguis, etc.—are responsible for tabes.

This latter notion is still widespread among the demi-mondaine and its devotees. It receives official sanction in many modern works on neurology and psychiatry, but, as may be gathered, it is one of the relics of the past, which contains psychological truth if not actual truth. The psychological truth is that venery is more apt to bring about syphilis, and syphilis has tabes as one of its aftermaths, but that venery itself should have the aftermath is not true.

Cold, Wet, and Exposure.—Cold, wet, and exposure are frequently invoked as agents contributing to the lessened resistance. They have nothing to do with tabes *per se*. In the presence of syphilis the role is very problematical. It is true that excessive marching may by its great fatigue bring an unsuspected tabes to the fore—just as an unexpected tabes may reveal itself in a sudden and apparently mysterious breaking of a bone; excessive exercise then acts to force a syndrome into recognition; it does not cause it.

Trauma.—Advocates for a traumatic origin for tabes may still be found, although it seems less and less likely with every advance in the pathology of the disease. Kurt Mendel, in a recent monograph (1910), discusses 11 cases in detail, while F. Schultze, in an analysis of 161 cases of tabes, discusses the possibility in 8. It is not unlikely that the accident and the tabes were coincidental. As to a further possibility, whether in a syphilitic a severe accident to the spine can determine the localization of a meningeal, inflammatory process, which in turn will bring about a tabes; this may be conceived in a positive sense when the trauma is evident and direct. It seems to have been established that so far as syphilis of the bones is concerned, and also for the localization of cerebral gummata, a traumatic experience may play some localizing role. Cerebral gummata, dural and osseous, are not infrequently found to develop at the site of a traumatic lesion. Possibly the same may be true for the meningeal precursors of a tabes.

After a tabetic process has been in progress, even if little apparent

to the patient, will trauma cause the process to advance more rapidly than it would without the trauma? In other words, is a tabes made worse by a trauma? Experience has answered this question in different ways. A reference to the section on the course of the disorder will show that no two cases ever develop in precisely the same manner. In some the progress is rapid and stormy; in others very insidious and long drawn out. No definite rule exists. It seems certain, for instance, that a blow on the abdomen has nothing to do with the development of a gastric crisis, nor a blow on the head with an optic atrophy. Furthermore, experience has repeatedly shown that one of the worst things for a tabetic, *i. e.*, particularly so far as his ataxia is concerned—is rest in bed. If this absolute quiet is, other things being equal, bad, it is difficult to grasp that a trauma can be a deleterious agent.

From the medico-legal point of view it is worthy of note that most accidents in question are the result of an already existing tabes, rather than the tabes being the result of the accident.

The whole question of whether a tabes is rendered worse by an accident can be answered only after a careful series of cytological studies of the cerebrospinal fluid have been made. At the present time it would appear that the activity of a syphilitic (tabetic, parietic) process may be more or less accurately estimated by the number of cells in the cerebrospinal fluid; higher counts indicating greater activity, lower counts diminished activity. In this way one may hope that the results of therapy may be controlled in a measure. So, also, the effects of trauma may be estimated. When a sufficiently large number of tabetic cases, before and after trauma, have been studied from this point of view, then and then only from our present-day knowledge can one state anything positive relative to the disadvantageous effects of trauma in tabes. No such studies are as yet available.

A correct estimation of the psychogenic element of financial hopes, of revenge, in making tabes worse after a trauma, especially in a litigated case, opens up another and extremely important subject. Although this aspect of the tabetic situation does not properly belong to a discussion on the factors of lowered resistance, yet it may be disposed of as well here as anywhere else.

In medico-legal interests the tabes and trauma question is an active one. Enough has been said to show that it is solely with this question of a tabes being made worse that discussion is pertinent. In litigated cases one rarely has an opportunity of estimating how badly off the patient was prior to the accident. Even assuming an honest belief on the part of a patient that he was not sick, or not sensibly inconvenienced by his malady, if he knew he had it, there are no known means of registering the grade of his incapacity previous to his accident, nor—it must be said—any certain rules to determine how badly off the accident has made him.

In estimating the psychic effect of shock one can only put forward a personal opinion. It is not a negligible factor. The shock of an accident may have no appreciable effect upon the syphilitic process

per se—concerning this, as stated, there are no available data, but there is little question that, litigation or no litigation, severe shock may bring about a very unfortunate mental attitude which incapacitates the patient very considerably, and usually for some time. In the presence of litigation such inability is invariably exaggerated, but such an exaggeration does not spring entirely from a false foundation. The instincts of self-protection will grasp every opportunity for furthering their cause, consciously as well as unconsciously. It therefore becomes extremely difficult to put an accurate measure upon the exact state of the disability, and a just judgment can only be arrived at by a careful review of the entire question.

It should be taken for granted that (a) an accident cannot cause a true tabes. Certain rare crushing lesions of the posterior columns and posterior roots may cause pseudotablic syndromes. A cytological test will decide the nature of the process: (b) It is very frequent that the slight beginning disability of the tabetic is the real cause for accidents of a certain nature to occur to him: (c) Accidents of a mild grade are of minor importance in causing any sudden access of the disease and consequent disability: (d) Severe accident, largely by reason of the mental features, often causes severe grades of disability. A proper valuing of the effect of the accident is best brought about by considering it from the mental rather than from the neurological side. In spite of necessary aggravation, exaggeration, etc., a tabetic—other things being equal—is less able to stand the mental (moral) effects of an accident. In this sense the tabes reduces his resistance to the trauma—not the trauma to the tabes.

Edinger's Views on Development of Tabes.—Before closing the section on secondary factors, a word relative to Edinger's point of view of the development of tabes. His general thesis involves the conception of a specific energy. The different cells of the body are in a position of equilibrium; if the function of one is disturbed, it affects all. Should a cell or group of cells become weaker, then the neighboring cell or cells of relatively related function must take up their task, or if a certain series of functions are called upon continuously, then the nerve paths utilized in the activity of these functions fatigue or are used up. The so-called occupation neuroses offer to Edinger examples of his general hypothesis.

When a chronic poisoning enters into the situation, the same rule applies. The functional apparatus of maintaining one's equilibrium is constantly operative so long as one is standing, sitting, or walking, even to a certain extent in reclining, or even sleeping. It is because of the constant use to which the sensory pathways underlying coördination are put that Edinger assumes they are used up in the presence of a chronic syphilitic toxemia.

Edinger supports his hypothesis by stating that such syphilitic individuals who have to be on their feet continually—officers, engineers, coachmen, women who work in contrast to women who do not—are more often tabetic, and because of the overworked coördination paths.

A number of interesting illustrations have been gathered from the literature which seem to lend support to the hypothesis. Gastric crises are prominent in heavy and frequent eaters; clitoral crises in nymphomaniacal women; arthropathies in joints damaged in early year; incontinence in one with previous enuresis, etc. For Edinger the syndrome exists on the basis of the toxin, but its manifestations are due to those factors which would induce the wearing out of a particular set of sensory neurones.

If Edinger's point of view were accepted without reservation, much restriction of the activity of a syphilitic individual would result, with the probable development of a tabophobia. It does not seem worth while in the present uncertainty of our knowledge to accept the therapeutic implications of the Edinger hypothesis. In a statistical review of some three hundred cases of tabes, I can recall only one among sailors. Syphilis is very common among this class; the coördination fiber tracts are usually very busy on the ocean: this one particular sailor—or sailor cook—had an arthropathy, and other tabetic signs, but had never developed any ataxia. This is not the place to thresh out the worth of the Edinger hypothesis. Reference is made to it as not yet being sufficiently proved to found a prophylactic theory for tabes in a syphilitic. If a fairly reasonable relation between occupation and the outbreak of tabes could be established, it would be highly desirable to know it, and be guided therapeutically.

Almost the only definite hint is obtained from a study of certain native Arabian tribes of Africa. Here the researches of Rudin have shown that the syphilitic incidence is extremely high—75 per cent. of the population being syphilitic—yet tabes, as well as paresis, seems to be almost unknown. These Mohammedans lead a nomadic agricultural life. Syphilis of the skin and bones is frequent, of the nervous system rare. Alcoholism is restricted. The inference would seem to be that in order to avoid tabes one might lead the outdoor life and remain abstinent—but Rudin thinks that this has nothing to do with it. The whole problem is one of civilization, and the demands of modern social adaptation.

Direct Treatment of Pains.—This may be symptomatic or specific. We shall here consider the symptomatic treatment first because the patient demands instant relief, and specific therapy is slower—if efficient. Moreover, the treatment of the tabes as syphilis may best be considered in a final section which deals *en masse* with the entire series of phenomena collected in this section.

The symptomatic treatment of the pains may be comprised under the pharmacal, general, and surgical heads. It is to be emphasized that the means here spoken of have little or nothing to do with the attack upon the disease process. They are purely palliative, and must not be used to the exclusion of the general antisypilitic treatment to be discussed later.

Pharmacal Therapy.—Various analgesics are of value in controlling the pain of tabes. The diagnosis once made, effort should be made to

raise the mental resistance of the patient to pain in general, and to avoid morphine. The psychical stimulus to pain bearing is of great importance. Many tabetic patients becoming fearful of the continuous severe pains, become great cowards, and come to fear even the thought of pain. They lose their grit and resistance, which is a great asset even in controverting the advance of the disease itself. Once embarked on the morphine path, the downfall is usually very rapid, and hospital parasitism is the result. One has to throw a great deal of energy into such tabetic patients to keep them out of the moral doldrums.

When considering the various analgesics, one has to bear in mind that great variation in patients is usual, hence one should go through the list systematically, and empirically ascertain which combinations have the best effect. Then when this is found it may be recalled that it may soon lose its effect; or will be used to excess, with a loss of that mental resistance so important to cultivate and to keep up to pitch.

Pyramidon, antipyrine, phenacetin, aspirin are among the most valuable. These can be used alone, or better in combination. These are used in doses of from 5 to 10 grains (0.3 to 0.6 gm.) or even more with severe pains. In prescribing mixtures one should take into consideration the rapidity of action of the drug (usually due to rapid solubility) with early relief, with its protracted action (insolubility and slow excretion). Other analgesics, such as lactophenin, chinin, trigemin, anesthesin, analgen, exalgen, may be given a trial and the results carefully checked up. Standard works on pharmacology will give the needed information regarding solubility and dangers. In using this general group of analgesics those with the aniline radical are usually stronger analgesics, but they have a disadvantage in reducing the oxidizing capacity of the blood and hence possess danger in large doses—causing cyanosis, shortness of breath, and all the symptoms of diminished oxidation. The advantages of their powerful analgesic action must then be obtained from small doses—rarely over 5 grains—to which other analgesics can be added to advantage, thus continuing and prolonging the action. Thus:

R—Pyramidon	gm.	0/3	gr. v
Aspirin	gm.	0/6	gr. x
Every half-hour for 3 powders; repeat in three hours.			
R—Acetanilide	gm.	0 3	gr. v
Antipyrine	gm.	0 3	gr. v
Sodium salicylate	gm.	1 3	gr. xx
1 powder every half-hour for 3 powders; repeat in three hours.			
R—Pyramidon	gm.	0/2	gr. ii j
Acetanilide	gm.	0/2	gr. ii j
Phenacetin	gm.	0/6	gr. x
1 powder every half-hour for 3 powders; repeat in three hours.			
R—Pyramidon	gm.	0/3	gr. v
Phenacetin	gm.	0 6	gr. x
Pantopon	gm.	0 005	gr. $\frac{1}{2}$
1 powder every half-hour for 3 powders; repeat in three hours.			

Pantopon should be used only occasionally, or its place taken by codeine, heroin, dionin, or other morphine derivatives. If possible this group of narcotics should be avoided.

Gastric crises may require morphine. It is to be recalled that three types of crises are to be distinguished: in one the root zones of the upper dorsal are involved, in a second the root zones are the lower dorsal, while in a third the pneumogastric is implicated. In the latter there is active nausea and vomiting, but little pain.

The morphine habit can be engendered in these gastric cases, but it seems less likely because of the comparative infrequency of the attacks. The analgesics noted may be employed. It is more or less futile to apply local gastric remedies. The trouble is not in the stomach, it is in the dorsal roots of the sensory nerves.

Cracked ice, cocaine, chloroform solutions, iodine, cerium oxalate, orexin, have all been recommended, but it is seriously to be questioned if any action upon the mucous membranes has any value. Possibly cocaine-orexin or other direct sensory narcotic may mitigate the severity of the pains, but it does not appear how, in view of our knowledge concerning the pathology of the pain. The general analgesics mentioned are more valuable; they may have to be given in suppository form, and then must be given in larger doses.

Hyoscyamine, aconitine, cannabis indica, atropine, gelsemine, may be added either by injection or by suppository. Certain patients are relieved by atropine as well as by morphine. In such cases the size of the dose of morphine may be diminished.

Local application of sprays, medicated vapors, local anesthetics, orthoform, chloroform, ether, etc., may be tried in laryngeal crises. Here again one must recall that the disease is not in or of the larynx; the cervical and medullary roots are at fault, and the general analgesics are to be relied on more and more.

Rectal, vesical, ureteral, urethral, kidney, and other crises may be met with the same general remedies. Local applications sometimes offer relief, but are usually disappointing.

It is not an infrequent experience to find that, failing to obtain relief by local applications, meddlesome surgery is invoked to the disadvantage of the entire situation. A careful and minute sensory investigation might have revealed the cause of the discomfort, and then a fairly adequate therapy invoked.

In the final summing up of efficient control of the pains and crises, it is to be remembered that each attack of pain, or each visceral crisis is a law unto itself. Some will be very mild, others severe; hence, in all cases the patient should have a graded series of reliefs. Those with a background of strong, resolute self-control will avoid the use of morphine until the more specific types of therapy have been of some aid in the situation.

I do not mention silver nitrate, ergot, protargol, nux vomica, organic extracts, etc., because they are of no service, nor is there any reason why they should be. That certain attacks have seemed to be helped

by this or that remedy may be easily explained on the ground of the great variability in the severity of different attacks, and not improbably the suggestive and tonic action of the thought that at last the doctor has obtained the wished for specific. One's tonic psychotherapy must be employed, not in connection with pharmaceutical or physical remedies—in the sense of being in any way vitally associated with such remedies—but must be a thing apart. The value of the remedies must be placed on their proper plane. The psychotherapy must be real and sincere, not a cheap and gaudy optimism.

Hydrotherapy and Massage.—It is by no means certain what effects baths have upon the tabetic process. Empirically many patients are benefited by a proper course of hydrotherapy. In many cases it is psychotherapy masquerading in the guise of hydrotherapy. In others it would appear that the baths themselves are of service. One thing is certain, however, no special bathing resort, and no special method of hydrotherpay is curative. The literature sent out from many bathing resorts as well as the articles written by physicians residing at such resorts, are often fulsome in praise of this or that bath or procedure, but they must be taken *cum grano salis*.

Viewed from the standpoint of the tabetic, however, the situation is largely an economic one. One of the chief functions of a physician who is called upon to treat a patient with tabes is to adjust the patient's expenditure as far as possible to his income. To encourage expensive trips to foreign baths, or to places at home which are of no special value, works an economic wrong to an individual who in time is apt to suffer greatly from lack of early forethought. One should therefore develop a home hydrotherapy even for well-to-do patients.

Hydrotherapy is used to relieve pains, and to encourage general metabolism. For pain, one employs hot water bottles, hot sand, electric heaters, hot packs, etc. It has been taught for years that extremes of heat are harmful. This is doubtful. Certainly short local hot applications for pain are not disadvantageous. Continuous hot baths, over 97° F., may be, but short time applications are not.

For gastric crises hot packs are often serviceable; hot sitz baths are useful in vesical, rectal, and other lower sacral root crises.

Much importance is laid upon the mineral or other constituent of the bath. Suphur, iron, arsenic, radioactive substances, pine needles, etc.—these are all descendants of magic, and intrinsically are worthless. They are more often the appanage of the charlatan, and are misused for their psychic effects. This is not a valid type of psychotherapy.

Heat and moisture are the desirable features. They may be applied in the form of water, plain or aërated (carbonated, etc., for skin stimuli), of packs, of mud, etc. There is no special merit in one type or another; yet special bath resorts develop a technique of application that has its own rules and regulations, and which have their individual psychological value.

All of the rules, regulations, forms, special appliances, and the general

technique of a well-regulated hydrotherapeutic establishment, such as are to be found in Virginia Hot Springs, Mudlavia, Mt. Clemons, Saratoga, Arkansas Hot Springs, etc., or in the establishments of France (Lamellou) and Germany (Nauheim, etc.) have their great value in the psychological principles which govern such institutions. With proper antisyphilitic therapy they are advantageous.

The regulation of the life, exercise, interests, etc., is of the greatest importance. No special rules can be laid down. Each patient is a law unto himself. (See Hinsdale's *Baths of the United States*.)

At home the baths may be given by a nurse or attendant, or by a member of the family. A full warm bath at about 85° to 90° F., lasting well over ten minutes, should be followed by a brisk rub or a cold douche. Such baths may be used for a month and then discontinued for a month to be renewed. The use of skin irritants, salt, alcohol, bran, etc., has no special value so far as the tabes is concerned. The chief end and aim is to stimulate physical vigor. This results from the action of the baths upon the circulatory organs, upon the muscular metabolism, usually much diminished in tabes, and upon the blood-forming organs. The general metabolic stimulation is a useful adjuvant to that of psychotherapy.

Under all circumstances hydrotherapy must be carried out under medical supervision. It must not be left entirely to nurses or bath attendants. The general rule to follow is about as follows: The first bath should not extend over three to four minutes; they are then gradually lengthened to ten to fifteen minutes. One takes two baths on successive days, on the third day the bath is omitted. After each bath the patient should rest for a half to two and a half hours. The temperature should be warm—not hot. Fifteen to twenty baths should constitute a treatment, to be renewed after as long a pause between baths.

Mechanotherapy.—Massage in the form of light friction with a deeper kneading in the back regions is a useful aid to hydrotherapy. The chief object to be obtained from massage is to compensate in part for the diminished muscular activity induced by the ataxia. If the ataxia is not marked enough to eliminate all exercise, massage may be omitted. At all events, since early and severe fatigue is a frequent condition in tabetics, massage and exercise must be carefully regulated.

If there is excessive hyperesthesia of the skin, massage is often distressing. Light stroking of the skin may then be found of some aid in relieving the pains.

Suspension.—Suspension may be here considered as a general mechanotherapeutic method. It is an interesting illustration of a medical fad. It is not inconceivable that certain patients were benefited, temporarily, but that anything like cure could result does not appear rational in view of the general pathological conditions underlying the tabetic syndrome.

Various osteopathic practices must be considered here. Personal experience has shown some hideous results. There has been no relief

seen that could not be brought about by more intelligent massage or counterirritation.

Orthopedic methods require a short word. They were chiefly devised to stretch the nerves thought to be affected—chiefly the sciatic. This was a useless and often mischievous mode of treatment.

Various orthopedic corsets, having for their object a local suspension action, have been utilized—at times accompanied by some striking betterment in pain, girdle sensations, even crises. It would appear that psychotherapy rather than the corsets was responsible; especially as the former is very rational. In view of the pathology, the latter can hardly be supported.

Counterirritation.—This method of treatment, long in vogue, by means of seton, actual cautery, blisters, etc., has been largely abandoned. While it is recognized that it has no effect upon the general pathology, and should never be employed as a sole means of treating tabes, yet at times counterirritation may be of great service, with other remedies, in relieving the pains. An important general principle is to make one's applications over the root zones, which a careful sensory examination reveals to be those chiefly implicated. Under all circumstances it should not be forgotten that anesthetic areas may be present in the regions to be treated. One should therefore avoid any remedies which tend to destroy the already under-par, skin-trophic influences. Counterirritation over the peripheral distributions of the pain is nonsense. One desires to influence, if possible, a more or less chronic meningeal process located about the sensory root zones. The trouble is not at the periphery. Thus in certain taboparetics the pains are due to thalamic lesions. It is manifestly absurd to treat the local, peripheral projections.

Electricity.—The present-day position relative to the influence of electrical currents upon tabetic pains is difficult to summarize. Personal experience does not show that ordinary methods of using these currents is of any distinct service. Yet one finds that careful observers report useful results, and which they believe are due to other than purely psychical means.

In the earlier days of electrotherapeutic enthusiasm general galvanization of the spinal column was widely extolled. Special methods were devised. They have practically been abandoned by conscientious physicians. Special methods of galvanization of the vagus, of the solar plexus, and medulla were equally praised. They also have gone with other magic incantations.

The faradic brush is still widely recommended for the sharp, shooting pains, and in its more developed forms as Franklinization, d'Arsonvallization has many adherents. Nagelschmidt reports results so striking as to create doubts as to the possible diseases under consideration. It is largely because of errors in diagnosis that electrotherapeutic results need to be carefully scanned.

High frequency applications have been among the later claimants to the attention of the neurologists, and increasing experience tends

to show that in this particular type of application one can affect the deeper root areas and bring about relief of pain. These are instances probably of electrical anesthesia. They are not of any permanent value and do not affect the process causing the pains. Precisely similar results can come from analgesics, cocaineization, or alcoholization of the posterior roots. It is only a question of choice of method of producing a temporary anesthesia.

Radio-active substances, as well as various light therapies, have not fulfilled any of their expectancies.

Electrical therapy of the crises by applications over or within the stomach, the bladder, the vagina, or rectum is all illusory. The disease process is not located in these organs. Quackery alone can find justification for such procedures at the present stage of our knowledge.

In one important particular, and apparently in one alone, is electricity of distinct value in tabes. This is in relation to the treatment of the atrophies, which are not infrequent in this disorder.

Surgical Treatment of Pains and Crises.—Inasmuch as the pathological seat has been determined for the greater number of patients, it is natural that the posterior roots would be attacked by surgical procedures for the relief of pain. The evolutionary steps were naturally through endomedullary measures. At first spinal applications of cocaine, eucaine, stovaine, adrenalin were employed, but the more radical procedures of Förster offer something more permanent.

In the indefinite or non-localized pains of tabes it would appear hardly worth while to adopt such a radical means for relief, yet there are instances in which the suffering is so great that the procedure of section of the posterior roots affected seems justifiable.

The history of posterior root section for intractable pain began with Bennett and Dana's first suggestion in 1888. Its general application to neuralgias in general—not tabetic pains—has had a rich development at the hands of Horsley, Chipault, Munro, Hartley, Frazier, Kerr, and others.

O. Förster formulated the general procedure for the relief of gastric crises. This was by section of the posterior root, cutting off the irritation of the sensory-sympathetic gastro-intestinal fibers, which pass through these dorsal roots. In his earlier publications Förster fixed a series of indications from which he has more recently (1913) departed in some particulars. These earlier ideas were that only extreme cases should be operated. Such are those with marked inanition, or those with marked morphinism, or those whose existence is threatened by the crises.

At least as many roots must be sectioned as show disease by sensory testing, yet there are certain anomalies of distribution which indicate the possibilities of intermittent sectioning. The details for this have not yet been determined.

Up to the time of Singer's general summary (July, 1912), 38 operations were available, many of which have not been published in detail. In 26 per cent. of these death followed—not all as a necessary result of the

operation, however. In the main the pains were for the most part stopped, although a number of disagreeable by-effects were obtained—some necessary, others due to faulty surgical technique. The pain returned in a number after some months. This may be interpreted in a number of ways—the most direct being that either the proper roots, or the proper number of the same were not cut. In a few cases the results were so valuable that the patients could resume their occupations. With increasing experience the results have been better. The more exact observations by sensory tests made accurate localization more possible, and certain modifications of technique—particularly the Gulecke procedures—have made it possible to do away with the earlier laminectomy operations. These modifications have made the double-sided operation less of a danger and introduced more certainty into the situation. At the present time of writing the Gulecke operation promises to be an important technical advance.

It is too early to state what the ultimate fate of the dorsal root operation is to be. The subject is under active discussion, and is modifying rapidly. It must always come up for careful consideration for severe gastric crises, and the best available position ascertained from both the neurological and surgical standpoints. The literature to 1912 may be found in the Singer contribution.

Treatment for Ataxia—Here will be discussed the methods devised to minimize the disability due to the ataxia. A number of these methods have been advocated, but only a few have survived as showing results of sufficient value to warrant their full consideration.

Frenkel, working on Raymond's material at the Salpêtrière, devised a series of training procedures which utilized the compensatory mechanisms of the eye and vestibular mechanisms. It is well known that these are of great service in training the child to walk. The child must see, and keep from falling through information derived from the vestibular organs. The vestibulospinal tracts convey these necessary messages to the limbs.

In tabes a whole series of deep sensibility impressions are cut off. These are bound up with receptors for joint movements and muscular tension which ultimately develop into a coördination which permits the limbs to control time and space relations for the purpose of walking. The gradual formation of these coördinations requires the aid of the optic nerve, ocular movements, and the semicircular canals, particularly the last. In time the whole situation becomes an automatically responsive mechanism, requiring little conscious direction, so far as locomotion and the performance of simple acts are concerned. The pathological alteration in tabes, however, cuts off, in varying degrees in individual patients, the pathways from these particular receptors, so that the information regarding movements, tensions, and weights, etc., can no longer be utilized. Hence the varying degrees of uncertainty in motor acts and the loss of control in time and space management, on the part particularly of the legs for walking, and the arms for manual labor, writing, etc.

In the exercises devised then the general viewpoint is that one needs to reëducate the limbs (1) by means of the eyes, and (2) by utilizing to the full those pathways not completely blocked off from carrying sensations. These two methods are combined in actual practice.

Exercises in Reëducation.—Only a short sketch of these exercises can be given here. For a more complete summary the reader is referred to the work of Frenkel, and the most thorough study by O. Förster.

In practice one finds patients who have trouble in walking, but (1) who are able to walk about fairly freely without support, but in whom the walking is altered and often leads to falls; (2) those who need a cane, or the arm of a companion—maybe also a cane; (3) those who cannot walk, but can stand, and (4) those who can neither stand nor walk. No attempt will be made to outline modes of treatment for these groups. The general principles must suffice. It is one of repeated exercises, combined with maximum attention. Repetition enables the individual to differentiate stimuli of the minutest intensity. One's sensibility becomes so great that frequent repetition of the stimuli act with the same force as less frequent response even to much stronger stimuli. The tabetic starts out to specially train his muscles to deeds never before thought of as possible, and inasmuch as the lesions in his nervous system are rarely so widespread as to cut off all the needed pathways, there nearly always remain some few neurones whose efficiency can be cultivated to the utmost. Naturally the greater the extent of the blocking lesions, the greater are the difficulties to be overcome.

A careful sensory chart must be prepared for each patient, particularly with reference to his knowledge of joint movements, his knowledge of muscular tensions, weights, etc., and all the factors of deep as well as superficial sensibility, protopathic as well as epicritic. The limbs are to be tested separately, and the possibilities of movement explored. Then the systematic exercises are started and persisted in.

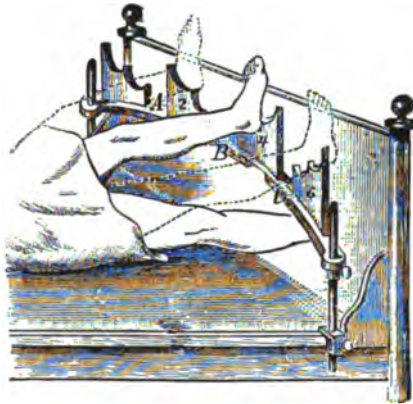
A hopeful stimulating attitude is necessary throughout the treatment, for by grit and determination bedridden tabetics, unable even to sit up in bed, have been so benefited by the exercises as to be able to walk down stairs backward. One's time and energy is thrown away on those morphinized individualities with fiber in them like a dish rag. Such are not worth treating as a rule—they become the prey of charlatans.

In normal walking there is a constant shift of the weight from one hip to another. In certain tabetics the loss of weight impression transmission makes this a serious difficulty to be overcome, and one of the most fundamental to be dealt with. It causes a difference in the kind of step of the individual. Short steps with dragging movements are common when this difficulty is present; whereas, the stamping long movements with falling forward is a mode of progression due to a different sensory loss.

In severe cases the exercises cannot begin with walking. They must either start with simple exercises on the back, or the simplest type of balancing exercises.

Lying upon the back with the lower extremities bare, one goes through a complete series of flexions, extensions, abductions, and adductions. These are carried out at first one leg at a time, later in unison, with eyes open and with eyes closed. When the muscular movements have acquired a certain ease and flexibility with some degree of precision—definite movements are carried out on the back position, such as putting the heel upon the opposite knee, or upon the opposite toe, or touching a series of posts which are placed upon the foot of the bed, or the feet are trained to rest in pocket-like bits of apparatus.

FIG. 23



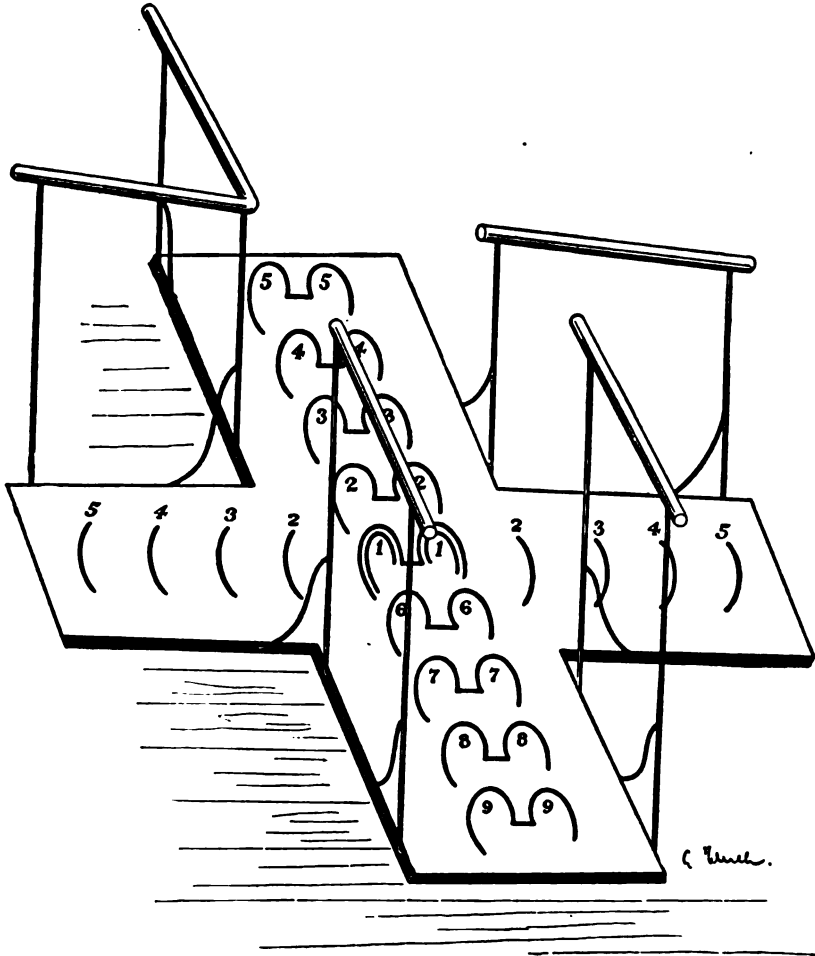
Bed apparatus for exercises.

When the legs and thighs have attained this efficiency—sitting up exercises are started. These consist in a series of movements of rising from a chair with the legs in varying positions, leaning forward and backward, sideways, rotating, etc. These exercises are all planned to obtain as full a control of the hip musculature as possible, for the control of the hip musculature is a very important feature in these exercises.

The walking movements then follow. These consist first in a series of side steps, or of steps about a circle. A series of footprints should be chalked out or painted upon a canvas sheet placed on the floor. The patient slowly revolves, placing his feet exactly upon the indicated footmark. In a similar manner sideways walking is practised, and movements of bending and of torsion follow. These are very important and are of the same nature as those practised in the sitting posture—save that here the patient stands up.

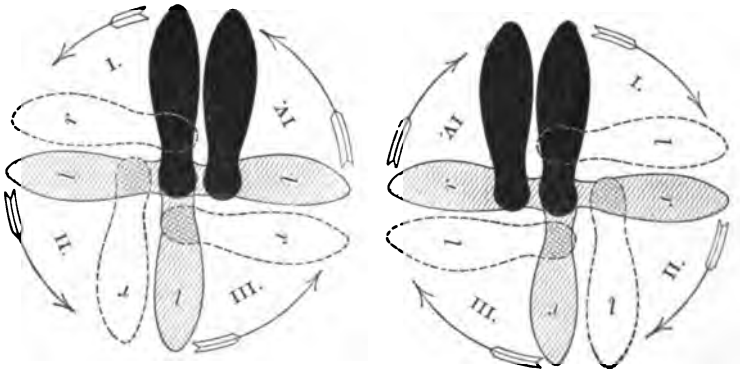
It is not necessary to have any elaborate apparatus for these exercises. The object of the whole procedure is to obtain precision in movement, and exercises which are elaborated to fit the individual case are superior to those which are more formal. When possible a useful motion is to be introduced. Thus in upper arm exercises typewriter practice is

FIG. 24



Practice board for patients unable to walk unassisted.

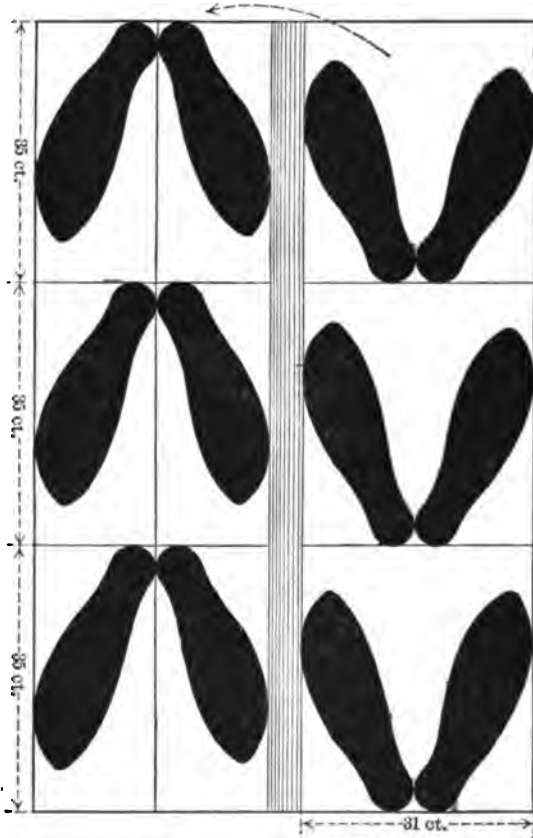
FIG. 25



Circle side-step movements in early stages of relearning to walk.

much more valuable than the use of the ordinary methods of having a patient stick his fingers on a series of pegs. Basket-weaving practice is better than more formal exercises. Rug-weaving on simple looms makes an excellent sitting position series of exercises. Blacksmithy work is excellent practice. One can carry on every desired movement, and be doing something useful at the same time.

FIG. 26



Footprints on floor for practice in walking

Many of the exercises may be combined with gymnastic appliances, weights, wands, dumb-bells, etc., not for the gymnastic part of it but as supplying a slight externalizing idea.

These exercises must be carefully regulated in duration. There is frequently a marked increase in the rapidity of the pulse. This must be watched, and when about 120 to 150 the patient must rest. Ten to twenty minute planned exercises are enough at first; then they are done twice a day, then gradually lengthened. In the beginning the control by the eyes plays a prominent role. But it is the ideal to do

away with the eye control and cultivate the sensibility. This can usually be done, and in such exercises the patient wears a mask.

FIG. 27



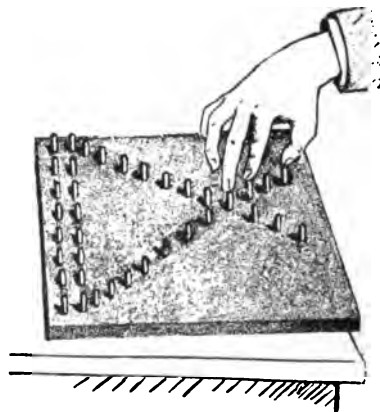
Zig-zag in floor for walking exercises.

FIG. 28



Ring exercises to train movements of limbs and aid in balancing.

FIG. 29



Exercises for upper extremity.

In view of the fact, already mentioned, that optic atrophy patients rarely have much ataxia, and more pertinently because it has been

observed that with increasing loss of eyesight tabetic patients often show diminishing ataxia, it appears very reasonable that the best way perhaps to train an ataxic is to reverse the Frenkel order and insist from the beginning on training the remnant of sensibility left and not depend upon the eyes. Recent experiences with this method seem to show that patients practising with a mask overcome their ataxia more rapidly than those who utilize their eyes. With either method the use of the stick in walking is to be avoided as much as possible. The patient may carry a cane to help him out in a dangerous situation, but in theory it should be over his shoulder or under his arm instead of being used as a support for the pelvis.

In training the upper extremities, exercises similar to those suggested for the lower limbs are devisable. They will consist in holding out the hands; movements of the fingers in definite directions; touching the thumb with the tips of the fingers (piano exercises are here good substitutes); movements of certain fingers, with holding of others; movements of individual phalanges; piling up of checkers or of round disks; the use of a board with holes in it to practise movements (knight's move tricks on a chess-board are good substitutes); buttoning and unbuttoning; opening and closing the fist; punching a punching bag; spoon exercises; writing exercises with chalk and blackboard; typewriter exercises; blind alphabet writing; drawing, etc.

These exercises are described at length in the works referred to, as well as others which may be desirable in certain rarer ataxias (speech, eye muscles, etc.). They should form a constituent part in the therapy of all those tabetics who have any desire to be able to get about. As for the lazy, indifferent hospital parasite little can be done; still it not infrequently happens that a little enthusiasm goes a long way in waking up a hopeless, hospital invalid to some degree of efficiency.

In certain cases of acutely progressing tabes the exercises are contra-indicated. Here absolute rest seems the only thing to be done, while a specific therapy is being used. Certain arthropathic cases do badly with much exercise. Here also the imminent danger of breaking bones is to be remembered. Tabetics with complicating muscular atrophy also do badly under exercises, and those patients with torn tendons are to be considered in the same light as the tabetic with arthropathies or fragile bones. Patients with syphilitic heart disease—not infrequent in tabes—are to be exercised with caution. Excessive adiposity, comparatively rare, also is a contraindication.

Etiological Treatment of Tabes.—Tabes is, in the highest degree probable, a syphilitic disease. Yet it has for the most part shown itself refractory to ordinary specific therapy. This is not due entirely to the fact that an early syphilis has been neglected—nor is it true that tabes is due to too much specific (Hg.) therapy, as has been claimed in times past. Patients with syphilis have been consistently well treated, and yet developed tabes; they have received no treatment at all and developed tabes, and they have been excessively treated and developed tabes. There are no convincing statistics to prove beyond

doubt which course most influences the later development of a tabes. (Nonne, loc. cit.)

At the same time there are certain considerations which tend to throw the whole question of tabes into line with what is known of many other disease reactions. In the first place the word tabes symbolizes a number of quite differing processes seen from the standpoint of development and causation. In certain individuals one can speak of a so-called metasyphilitic process in which the chief destructive element is of toxic nature. The neurone is attacked in a manner analogous to that seen in a post-diphtheritic paralysis. The invading organism may have disappeared entirely and yet its products act deleteriously. On the other hand, certain tabetic individuals show in their posterior roots definite gummatous infiltrations, which by their pressure alone are capable of inducing the destruction of the sensory neurones. These must certainly be viewed in an entirely different light therapeutically from the former ones. Then again, other tabetics have shown little spinal meningeal disease, but a syphilitic focus in some other part of the body, capable of liberating syphilitic toxins which may act upon the sensory neurones.

Furthermore, the serobiological tests seem to show, in the first place, no Wassermann reaction or 10 per cent. only with 0.2 of serum, but positive with larger amounts; an increase of cells in the fluid; a positive globulin reaction; hence, one must support the view that a syphilitic process is going on, and more or less actively when the cell count is large. It may here be recalled that Boas, of Copenhagen, has claimed that the presence of a blood Wassermann in a tabetic is good evidence of his having had no syphilitic treatment.

All of these considerations point to the desirability of a specific attack, leaving it to be patiently sought out why the treatment as now carried out for the initial syphilis does not prevent an after-coming tabes.

It seems highly probable, with the knowledge obtainable from the cerebrospinal fluid, that fewer and fewer cases of tabes will develop because of a more adequate treatment of the fundamental disease, *i. e.*, a treatment controlled by other than the heretofore empirical methods.

From this it follows that the earlier it is definitely learned that a tabetic process is in progress the more active should be the attack. It has not been shown as yet conclusively that the usually employed specific remedies do any great harm to the nervous system when administered in a fairly reasonable manner. An antisiphilitic treatment for certain patients with tabes—old cases with marked degenerative changes offers little hope of reward.

The extremely important subject of what has been termed "neuro-recidive" should be considered briefly at this time, since it is pertinent to the objection that specific therapy, not alone by the arsenic preparations—cacodylates, salvarsan, neosalvarsan, etc.,—but also by mercurial preparations, has been responsible for certain very definite

advances in syphilitic disease—optic atrophy, vestibular affections, acute excitements, epilepsies, apoplexies, etc. This subject has been discussed at greater length in speaking of cerebral syphilis (p. 337), syphilis of the base and the convexity, but it also has its application in any discussion on the specific therapy of tabes. As previously stated, it seems definitely shown that the so-called bad results of specific therapy (neurorecidive) are due, not to the remedies employed, but to the disease itself, and that a continuance of a proper medication clears them up. This has been shown to be true even in one of the most feared of all syphilitic results—optic nerve atrophy—by Spiethoff and others. Just what effect certain types of therapy (*i. e.*, salvarsan) has in bringing about an increase in local activity of endotoxin formation—the so-called Herxheimer reaction—is discussed in another place.

Salvarsan and Neosalvarsan in Tabes.—At no time in the history of the salvarsan movement has it been said that at last an efficient remedy has been found for tabes. In fact, there has been from the first a definite pessimistic attitude. This is explicable on the general ground that the treatment by salvarsan had first to be applied to the collection of old tabetics, of whom little was to be expected.

The results of therapy reported by Azna, Hammer, Geronne, Dunger, Oppenheim, and others, which were mostly negative, are largely to be explained on this ground. In much of the earlier literature it is impossible to learn what kind of patient was chosen, what stage of the disease, what was the clinical type of the disease, etc., all of these points being of much importance in arriving at a fair estimate of the therapeutic situation. Certain of the articles reporting bad results are so tinged with an *a priori*, definite, negativistic attitude toward all therapy that they are of no value in any judicial summing up.

Certain authors report that their patients have become distinctly worse, so far as pains, girdle sensations, etc., have been concerned. Oppenheim's early reports have been along this line particularly. While the details of his reports are not altogether satisfactory it is not improbable that some of the increase in pain has been due to the usual exciting action (Herxheimer) set up in the posterior roots by insufficient salvarsan therapy. If the attack had been energetically followed up the results would have been much better.

In more recent times, however, the opinion is gaining ground, and we believe validly, that the use of salvarsan, and its related products, is distinctly beneficial (Singer, Nonne, *loc. cit.*). It has been noted that tabes so far as its pathological features are concerned, may present a variety of trends, and it is becoming more and more apparent that salvarsan is valuable particularly in those patients where there is a comparatively high cellular count, and in whom irritative phenomena point to a more or less active inflammatory lesion of the sensory root zones. Here one finds much benefit following salvarsan therapy. Many such cases are found among early tabes, *i. e.*, those originating soon after the syphilitic infection. Good results are being reported in increasing numbers. The beginning pains are relieved, diminished

knee-jerks have come back, and bladder difficulties been much improved.

Salvarsan as well as the mercurial preparations have given striking results in those patients in whom the tabes is a result of a more definite cerebrospinal syphilis with marked cellular exudate, and strongly positive Wassermann in the fluid. Salvarsan has also been of value in that group of cases in which an accessory syphilitic focus exists in some other part of the body.

In all of these types the action of salvarsan upon the pains has been beneficial. This has not been found to be invariable by any means; in fact, the number of cases distinctly and permanently benefited is as yet comparatively small, but in well-chosen cases the good results are often very striking. The length of time that these chosen cases have been under observation is as yet too short to permit positive dicta, but in the types mentioned the results thus far obtained are hopeful. It must be recalled that similar results were obtained by the use of mercury, but personal convictions lead to the belief that better results have come from salvarsan in the treatment of tabetic pains than from mercury. This may be due to the fact that at the present time these patients are treated earlier and with greater vigor. The cytological tests have provided a means not only for earlier diagnosis, but also for clearer indications as to the extent of the therapy. With more energetic and massive salvarsan therapy, given until a modified Wassermann results, the present indications are that one has in salvarsan a valuable remedy, not only to cut short the process going on, but to definitely kill all active, toxic producing agents.

The details of salvarsan and neosalvarsan therapy are given in these volumes in a special chapter written by Captain Nichols, and are not repeated here. From six to eight injections of 0.6 gram salvarsan within six to eight weeks is not too severe therapy if the tabes seems to be one accompanied by the frank expression of an active, meningeal, inflammatory reaction (pleocytosis, etc.), with pains, with possible ocular palsies or active crises with definite sensory skin accompaniments. Such doses are also justified in those patients with tabes coming on early, *i. e.*, within five years of infection, and also in those patients with probable syphilitic foci outside of the nervous system. The chief end and aim of the specific salvarsan therapy is to kill the organisms, and as rapidly and as completely as possible. In all tabetic patients in whom it seems probable that a fairly active process is in progress such direct attack upon the organism is advisable.

It is in the old, long-standing syphilitics, who slowly develop the tabetic syndrome, with low cellular count and questionable or absent Wassermann in blood or fluid, that the poorest results have thus far been obtained. Some of these are absolutely refractory.

The treatment of tabetic pains is only a part of the general question of the treatment of tabes as a whole. Passing to this broader subject, a *resume* of the present-day situation is desirable. It is here seen that the salvarsan treatment of tabes has rendered increasingly satisfactory

service as it has been more definitely grasped that many early failures were in large part due to insufficient and faint-hearted therapy. The tendency has been toward the opinion that more intense therapy gives better results, and it is apparently more and more important to consider combination forms of therapy of increasing efficiency in tabes, and general paresis as well. This latter subject is discussed separately.

Before considering the increasing evidence of the value of salvarsan, and particularly of salvarsan combined with mercury, controlled by lumbar puncture and cytobiological examinations, it needs to be restated that benign tabes is not unknown. Certain tabetic patients get to a very definite stationary period apart from intrinsic therapy. Yet such patients are comparatively rare. Furthermore, while discussing the results of salvarsan therapy, it is to be recollected that good results have not been unknown from mercurial therapy. Thus, the use of calomel, bichloride, salicylates, enesol, and other mercurial combinations has been followed by good results in many instances. These results should emphasize the ever to be remembered fact that individualization of the therapy is desirable.

The salvarsan therapy of tabes, however, has offered more certain, more rapid, and more definite results. Some of these, so far as the tabetic syndrome is concerned, remain to be considered.

The pains have been dealt with at length. The ataxia in a number of patients has been much improved. Inasmuch as this particular difficulty often yields very readily to other types of therapy—exercise, psychotherapy—it affords a less valuable criterion as to the value of salvarsan than it otherwise would.

The Argyll-Robertson pupil has been known to disappear in both tabes and paresis, following salvarsan therapy. Pupillary inequalities have been cleared up, and in some instances optic atrophy has been benefited. Optic atrophy as a neurorecidive seems to be fairly well shown to be a product of faint-hearted, salvarsan therapy.

A further word of explanation to account for certain features of the neurorecidive in general seems pertinent here. Recent studies in tabes and in paresis have shown following an initial dose of salvarsan, gm. 0.2 to 0.4, that a previously negative Wassermann in the blood has become positive. It seems justifiable to believe that a dormant syphilitic focus has become activated, and that previously dormant spirochetes have been stimulated into activity. Nichols and Hough in fact report the finding of active spirochetes in the spinal fluid of a patient with cerebrospinal syphilis in the early second stage. This patient was treated by salvarsan and there resulted the typical picture of a neurorecidive with a positive Wassermann, and active pleocytosis, and the picture of a confusional excitement with aphasic and hemiparetic signs.

Following the neurorecidive, inoculation experiments with the cerebrospinal fluid resulted in obtaining the organisms in large quantities from the testicle of the rabbit. Subsequent treatment by salvarsan and mercury caused a fall in the cerebrospinal pleocytosis, and a marked improvement of the patient from his neurorecidive. Here the proof

seems fairly clear that whereas the salvarsan possibly activated a syphilitic focus, yet the symptoms were due to a progression of the syphilitic process. This process was fully controlled by later therapy.

It thus seems probable that in that type of tabes with low-grade radiculitis, with accessory syphilitic foci, that an activation may take place following an initial salvarsan or neosalvarsan injection. Toxins are then thrown out, optic nerve poisoning advances, but if the initial therapy is persisted in, the retinal changes will diminish and finally may be arrested. The atrophy is not therefore due to the arsenic.

Ocular palsies have been cleared up after salvarsan, facial palsies as well.

FIG. 30

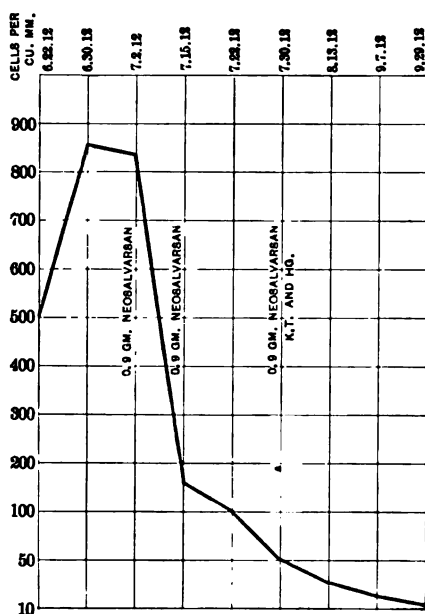


Chart illustrating the fall in the number of cerebrospinal cells following salvarsan therapy in cerebrospinal syphilis. (Nichols and Hough.)

Thus far the following symptoms have shown improvement: Pain crises, ataxia, external and internal ocular palsies, bladder and rectal weakness, impotence, lost or diminished knee-jerks, facial palsy, optic atrophy, loss of memory, loss of appetite, general loss of strength and vigor. At the same time it must be stated that all of these symptoms have been noted to increase in severity.

Of recent optimistic expressions, those of Dreyfuss, working in Frankfurt, are the most encouraging, and are more in line with the general trend of opinion at the present time of writing. Among other things he says that although the results of the treatment of tabes and paresis do not permit positive conclusions, yet they point in a hopeful direction.

The differences in results reported—and they are many—are due largely to the methods of treatment employed.

An insufficient treatment of tabes—*i. e.*, 1.5 gram of salvarsan or its neosalvarsan equivalent—either has no influence upon tabes, or it makes it worse. Should those patients be more energetically treated, they show marked improvement, even when the initial treatment has resulted in a definite regression of the patient's condition. Many patients cannot take combined salvarsan and mercurial treatment, although other things being equal a combined treatment will give the best results.

An outline of such an energetic combined treatment for tabes—and for paresis—is as follows:

First day	0.03 calomel, hypodermically	
Third day	0.05 calomel, hypodermically	
Fifth day	0.4 salvarsan, intravenously	Neo-salvarsan, dose iv
Seventh day	0.5 salvarsan, intravenously	Neo-salvarsan, dose v
Ninth day	0.05 calomel, hypodermically	
Eleventh day	0.05 calomel, hypodermically	
Thirteenth day	0.4 salvarsan, intravenously	Neo-salvarsan, dose iv
Fifteenth day	0.5 salvarsan, intravenously	Neo-salvarsan, dose v
Seventeenth day	0.05 calomel, hypodermically	

This should be kept up for a six weeks' cure with careful watching of the fluid and the general condition of the patient, until about twelve calomel injections are given, and 5.4 grams of salvarsan.

This is only a scheme showing the general requirements. Individualization is very important. Certain patients can follow out such an intense course, in others one must go more slowly. Certain patients do not bear calomel. In such an event the salicylate or enesol may be substituted. Naturally the absolute technique of the salvarsan infusions must be followed out, one of the chief requirements being the freshly distilled water.

A definite rise in temperature is a contraindication of the carrying out of too energetic a program. Influenza-like symptoms, with general malaise, slight bronchitis, constipation are definite contraindications. Patients should rest in bed at least a day or two after the salvarsan infusions. In fact, in following out such a cure rest in bed is highly desirable.

One series of six to eight weeks is not always sufficient. It should be repeated after three or four months or more. Dreyfuss notes that if at least 5 or 6 grams of salvarsan cannot be used it were better not to use salvarsan at all.

The Swift-Ellis method, already outlined, has been more thoroughly tried out in tabes than in any other type of nervous syphilis. Here the results have been more gratifying than in any other form. Whether this seems so by reason of the slow progress of tabes cannot as yet be determined, but the action of the therapy upon the serobiological reactions are so striking as to demand the earnest attention of all. We here append some of the results obtained in some of Swift and Ellis' cases.

History of F. H. P., aged forty-seven years; tabes dorsalis, duration fourteen months; primary syphilis, eight years ago.

Date.	Blood.	Cerebrospinal fluid.			Treatment.		Clinical condition.		
		Wassermann reaction.	Cells.	Noguchi globulin.	Wassermann reaction.			Intravenous.	Intraspinalous.
					Liver. antigen.	Heart cholesterolin antigen.			
1913									
Jan. 17	++	225	++	0.05 c.c. ++	0.05 c.c. ++				
Jan. 22	0.75 gm. neosalvarsan.			
Jan. 28	++	0.6 gm. neosalvarsan			
Feb. 4, 5	++	125	+	0.05 c.c. ++	0.05 c.c. ++	0.75 gm. neosalvarsan	30 c.c. 40% serum		
Feb. 11, 12	++	56	+	0.05 c.c. ++	0.05 c.c. ++	0.75 gm. neosalvarsan	30 c.c. 40% serum	Stationary.	
Feb. 24, 25	++	30	0.05 c.c. ++	0.05 c.c. ++	0.75 gm. neosalvarsan	30 c.c. 40% serum		

History No. 893. F. B.; tabes dorsalis, duration two years; syphilis, thirteen years ago.

Date.	Blood.	Cerebrospinal fluid.				Treatment.		Clinical condition.
		Cells.	Noguchi globulin.	Wassermann reaction.		Intravenous.	Intraspinalous.	
				Liver antigen.	Heart antigen.			
1912								
Oct. 18	+	40	±	0.5 c.c. +	0.75 gm. neosalvarsan		
Oct. 28	++	0.45 gm. neosalvarsan		
Nov. 2	57	+	0.5 c.c. + ±			
Nov. 4	++	47	+	0.5 c.c. ++	0.9 gm. neosalvarsan	30 c.c. 40% serum	
Nov. 12	13	+	0.5 c.c. —	0.9 gm. neosalvarsan	30 c.c. 40% serum	
Nov. 18	0.9 gm. neosalvarsan		
Nov. 25	+ ±	0.9 gm. neosalvarsan		
Dec. 2	+ ±	10	+	0.5 c.c. —	0.9 gm. neosalvarsan	30 c.c. 40% serum	
Dec. 16	0.9 gm. neosalvarsan		
Dec. 17	2	±	0.5 c.c. —			
1913								
Jan. 25	7	±	0.5 c.c. —			
Feb. 8	++	5	±	0.5 c.c. —	0.9 gm. neosalvarsan	30 c.c. 40% serum	Improved.

History No. 675. H. G. G., aged forty-two years; tabes dorsalis, duration two and one-half years; syphilis denied.

Date.	Blood.		Cerebrospinal fluid.			Treatment.		Clinical condition.
	Wassermann reaction.	Cells.	Noguchi globulin.	Wassermann reaction. Liver antigen.	Heart antigen.	Intravenous.	Intraspinal.	
1912 Mar. 18	±	35	±	0.3 c.c. ++				
May 2	—							
May 5	41	+	0.3 c.c. ++	30 c.c. 40% serum	
May 14	24	±	0.4 c.c. ++	30 c.c. 40% serum	
May 21	17	±	0.4 c.c. ++	30 c.c. 40% serum	
June 10	—							
June 20	14	±	0.5 c.c. ++	30 c.c. 40% serum	
June 27	33	±	0.5 c.c. ++	30 c.c. 40% serum	
July 9	12	±	0.5 c.c. ++	30 c.c. 40% serum	
July 21	35	+	0.5 c.c. ++	30 c.c. 40% serum	
July 30	—							
Sept. 19	14	—	0.5 c.c. ±	30 c.c. 40% serum	
Oct. 1	8	—	0.5 c.c. —	30 c.c. 40% serum	
Oct. 19	7	—	0.5 c.c. —	30 c.c. 40% serum	Much improved.
1913 Jan. 5	5	—	0.5 c.c. —	0.5 c.c. —	Died of lobar pneumonia.

EFFECT OF TREATMENT ON BLOOD AND SPINAL FLUID

On admission, tabes dorsalis.				On date of last lumbar puncture.										
Case No.	Date.	Blood.			Cerebrospinal fluid.			Date.	Blood.			Cerebrospinal fluid.		
		Wasser- mann reaction	Cell count.	Noguchi globulin	Wassermann reaction.	Liver antigen.	Heart cholesterin antigen.		Wasser- mann reaction	Cell count	Noguchi globulin	Wassermann reaction.	Liver antigen.	Heart cholesterin antigen.
80 B. A.	Feb. 12, 1911	—	8	±	0.1 c.c. —	Oct. 21, 1911	—	6	+	0.1 c.c. —			
152 O. F.	Apr. 4, 1911	++	27	++	0.1 c.c. —	Feb. 15, 1913	±	28	±	0.3 c.c. ++	0.2 c.c. ++	+	
555 P. J. F.	Feb. 8, 1912	—	14	+	0.5 c.c. —	Nov. 12, 1912	—	3	—	0.5 c.c. —			
557 C. H. L.	Oct. 25, 1911	—	42	++	0.1 c.c. ++	Sept. 24, 1912	—	8	—	0.5 c.c. —			
836 O. J. L.	Sept. 19, 1912	—	45	±	0.5 c.c. —	Nov. 12, 1912	—	8	—	0.5 c.c. —			
485 C. M.	Jan. 22, 1912	—	130	++	0.5 c.c. ++	Nov. 19, 1912	—	4	+	0.5 c.c. —			
85 C. L. V.	Jan. 16, 1911	±	125	++	0.1 c.c. —	Sept. 10, 1912	—	1	—	0.5 c.c. —			
213 O. W. A.	June 13, 1911	++	110	—	0.1 c.c. ++	Feb. 22, 1913	++	1	—	0.4 c.c. ++	0.3 c.c. ++	+	
559 J. J. S.	Jan. 22, 1912	+	30	+	0.5 c.c. +	Oct. 1, 1912	—	6	±	0.5 c.c. —			
893 F. B.	Oct. 18, 1912	+	40	±	0.5 c.c. +	Feb. 8, 1913	++	5	+	0.5 c.c. —	0.5 c.c. —		
113 W. L. S.	Mar. 7, 1911	++	72	++	0.1 c.c. —	Nov. 23, 1912	++	6	±	0.5 c.c. —			
517 W. B.	Jan. 25, 1912	—	55	+	0.5 c.c. ±	Nov. 12, 1912	—	3	—	0.5 c.c. —			
674 B. d'H.	Jan. 24, 1912	++	55	+	0.2 c.c. ++	July 16, 1912	±	21	±	0.2 c.c. ++	0.5 c.c. —		
675 H. G. G.	Mar. 18, 1912	—	35	±	0.3 c.c. ++	Jan. 5, 1913	—	5	—	0.5 c.c. —	0.5 c.c. —		

Italics: Patients treated by intravenous injections of salvarsan alone. Roman: Patients treated by intravenous and intraspinal method. Heavy Face: Patients treated by intraspinal injections of foreign sera.

The history of case F. H. P., on page 400, shows a remarkable active result obtained within six weeks.

The history of case F. B., on page 401, shows a closely related result. In both, intravenous and intraspinal therapy was applied. In both cases the good results appear more strikingly following the intraspinal injections.

H. G. G. (see table on page 402), treated only by the intraspinal method, showed excellent results. His death from pneumonia will permit important researches to be made.

Finally, I am enabled to present the results obtained in the treatment of 14 patients (see table on page 403) by the methods outlined.

Other Specific Therapy for Tabes.—In a very suggestive paper on the modern treatment of syphilis, Neisser makes the remark that he did not intend to break his head trying to decide whether salvarsan was better than other methods of treating syphilis. Some of the good results obtained by the use of salvarsan in the treatment of tabes, sketched in the preceding paragraphs, it may be recalled had also been obtained in many cases by the use of mercury. In a few instances one spoke of the arrest of the tabetic processes, even if it could not be claimed that a cure had resulted. Spontaneous remissions in tabes have been known for years, and yet in the main the results of mercurial therapy in tabes have been disappointing. It will therefore be years before a final decision can be reached relative to the value of salvarsan in the treatment of tabes.

There are contraindications to the use of salvarsan—these are the inconveniences, and at the present time a full energetic course of salvarsan therapy as outlined may be an economic burden to many patients. Furthermore, as has been pointed out, the present belief is that arsenic and mercury combined have a better action in tabes than either alone. It is therefore desirable to outline certain methods of using mercury in the treatment of tabes.

It is recognized that neither arsenic nor mercury are of any particular service in restoring degenerated nervous tissue. Their action is chiefly toxic to the syphilitic organism. They are both spirochetal poisons, the advantage seeming to lie with salvarsan in the rapidity of its action. In nervous syphilis other than tabes, its action is certainly more rapidly seen, and its good as well as bad effects sooner apparent. Yet mercury is a powerful poison to the syphilis organism. But arsenic has a very definite action upon the general metabolism of the body which is largely missing in mercury, and it has certain claims to preference by reason of this accessory value.

A large number of mercurial preparations are available. Neisser is of the opinion that hypodermic therapy is by far the best; yet there are not wanting those who prefer inunction or oral medication. The injection method has the great advantage of more accurate dosage, it being almost impossible to determine by inunction or oral medication how much mercury is being taken in. This is particularly true for pill medication. The use of a comparatively large amount of mercury in

a short time is the ideal mode of killing the parasite and is the method to be aimed at. In all cases the medication must be checked by cytological and serological examinations. Many patients with tabes and showing a negative blood reaction before the beginning of an intensive therapy, suddenly show a positive reaction after the mercury. These patients are among those who appear to offer good results.

Inunction Treatment.—Erb has repeatedly been an advocate of the method by mercurial inunctions. Sarbo in more recent times has recommended the inunctions, especially in the treatment of the eye palsies.

Three or 4 grams of unguentum hydrargyri are used daily until 30 to 40 grams are used. One can follow the usual routine of six days' inunction with one day hot bath. If this dosage is well borne the administration of ointment is continued until 120 to 160 grams are used. Following this, potassium iodide in 1 to 1½ gram doses daily is used for six to eight weeks, when a new course of inunctions, using 120 to 160 grams of the ointment, is given. Hydrotherapy may be intermittently given, or be reserved until the end of the inunctions.

There is little question that the results obtained in certain patients with tabes, by the inunction treatment, are encouraging, but many of the most promising show relapses.

Salicylates.—Salicylate preparations have their advocates. A number are on the market. Enesol is one of the most convenient, although the galenical salicylate is as efficacious. Schaffer (loc. cit.) recommends enesol highly. A number of excellent results have been obtained by the early and energetic use of the salicylate preparations in ocular palsies, loss of knee-jerks, bladder disturbances, ataxia, gastric crises, cutting pains.

The salicylates (enesol, lin; salicarsenat) are given in doses of 2 c.c. (0.06 gram) every second day for 20 to 30 doses. They are administered hypodermically, intramuscularly, usually in the thigh or buttocks. One must be on one's guard against abscess formation by using the strictest asepsis and the best materials.

Other soluble salts are the biniodide, much praised by Fournier, the benzoate, the bichloride, and the cyanide. I believe them to be inferior to the salicylates in general, but idiosyncrasies often arise whereby one preparation is not as well borne as another.

As the hypodermic methods have been described in detail elsewhere in the treatment of cerebral syphilis (p. 332) I shall not repeat them here.

Epidural and Subdural Injections.—The clearer appreciation that a meningeal process in the posterior root areas is responsible for the pains in tabes has led to a direct attack by epidural and subdural injections. Mercurial preparations (biniodide) were injected directly into the subdural spaces. Collargol was also used in a similar manner. Fibrolysin was also employed. Electromercural gave surprising results according to Mestrezath and Sappey. The mercury was thought to act directly upon the posterior roots, inducing a compensatory

meningeal irritation. There results at first an increase in the pains with later subsidence. The method is still too new to decide upon.

Sicard and Cathelin advise epidural injections acting upon the nerve roots in the sacral canal. Cocaine in 10 per cent. and stovaine in 4 per cent. solutions are employed. These proceedings are preliminary to the more radical or Förster operations.

The treatment for arthropathies is surgical. Other features of the therapy will be discussed under "general management," following the section on spinal syphilis.

7. SYPHILITIC PSYCHOSES

Here will be discussed forms of mental disturbance other than paresis, and also those which have not been specifically dealt with, as such, in the section on cerebral syphilis.

The anatomical researches of Alzheimer, the cytological work of Plaut, not to quote the many others, have tended to bring into the foreground other forms of mental disturbance—mostly of an acute or subacute character, conditioned by the syphilitic virus, and yet which do not comport in general to the mental disturbances already outlined.

Forms.—Neurasthenia.—Kraepelin speaks of a syphilitic neurasthenia—a form of mental disturbance much written upon by earlier authors. We are not speaking of the preneurasthenic phase of a cerebral syphilis or of a paresis. It is apt to appear shortly after infection and manifest itself in a nervous discomfort, difficulty in thinking, irritability, disturbance of sleep, pressure in the head, variable and changeable discomfort and pain. To these may be added slight depression, dizziness, confusion, anxiety, slight difficulty in finding words, temperature variations, paresthesiæ, and nausea.

Many students prefer to interpret these symptoms as a direct result of the infection, and not as a circumscribed syndrome; but slight vascular changes, minute pupillary alterations, and particularly evidence of meningeal irritation as shown by the spinal-fluid lymphocytosis, point in the direction of its being something more than a simple infectious reaction.

Pseudoparesis.—A common mental picture is that of syphilitic pseudoparesis. This has already been described.

Delirious Confusion.—A somewhat related picture is that form emphasized by Marcus under the title of a delirious confusion. It may come on in the first year after infection or much later. The onset is usually rapid, there is much psychomotor activity, insomnia, and anxiety. Confusion for persons and objects, disorientation in time and place and various auditory, optic, and haptic hallucinations are present. The patients are much frightened, often greatly excited, homicidal or suicidal.

At times there is little to call attention to a syphilitic etiology; with some, however, cranial nerve affections point to the causative factor.

PLATE XXXVI

Fig. 1



**Brain of a Patient with a Syphilitic Psychosis of Acute Maniacal Type.
Enlarged and Engorged Vessels. Syphilitic Meningitis.**

Fig. 2



**Brain of a Patient with a Chronic Syphilitic Psychosis of Maniacal Type.
Syphilitic Leptomeningitis, Pachymeningitis.**

Certain confusional patients develop a typical Korsakow syndrome. There is no polyneuritis as a rule. There is disorientation, loss of attention, amnesia, confabulation, with periodic somnolent attacks.

Psychosis Simulating Manic-depressive Type.—Westphal has called attention to a small group of patients whose psychoses resemble those of the manic-depressive type. In the manic phase they resemble the more lucid expansive paretics. A slight depression ushers in a mild euphoria, with unrest, irritability, slight lapses in memory, with ideas of grandeur and hallucinations, particularly of hearing, often persecuting in character. Finck has studied the false manic-depressives, and comments on the frequency of hallucinations of hearing.

Ballet has described depressed forms, often, however, premonitory to paresis.

Plaut has shown, by means of the cytological, serological tests, the syphilitic nature of these forms of mild meningitis and has differentiated them from paresis largely by reason of the high lymphocytosis, and negative fluid Wassermann.

Psychoses Simulating Paranoid Types of Dementia Præcox.—A certain number of syphilitic psychoses have come under observation whose clinical picture has closely resembled the paranoid types of dementia præcox. Hebephrenic and catatonic resemblances have also been described (Ilberg, Westphal). These forms present a number of extremely difficult diagnostic problems. Hallucinations, chiefly of hearing, and delusions of persecution occupy a predominant position in the clinical picture, physical signs being minimal or absent. A characteristic slow development takes place with moodiness, irritability, anxiety, and suspiciousness. Ideas of jealousy and of persecution are frequent. The patients believe themselves to be watched on the streets, are insulted, made fun of, or sworn at. They are spied upon, cheated, attempts are made to poison them. The hallucinatory voices come over the telephone, from the police, from within their own bodies, from beneath the bed. Optical pictures may be hallucinated as well, and more of the patients feel sick: they have "hypochondriasis." Occasionally ideas of sinfulness are expressed, and in some, ideas of grandeur of the type seen in paranoid types of dementia præcox. Consciousness is usually uninvolved, orientation unimpaired, or occasional confusional periods may lead to mistakes in objects and persons of the environment. Memory may be perfect, or at times slightly impaired. Again, there is much memory defect.

The emotional state varies greatly in this group. Kraepelin describes them as depressed, crying, anxious, quiet, irritable, busy, quarrelsome, self-conscious, or euphoric. On the whole, there is a certain blunting of the sensibilities with indifference or lack of interest in the situation. The general impression is, for the most part, of superficiality. Occasionally violent, impulsive activity shows itself in a manner quite similar to that seen in hebephrenics. Mild attacks of fainting, dizziness, transitory aphasia, mild epileptiform lapses, not unlike those seen in schizophrenia, have been recorded for this group of patients.

The conduct mirrors the emotional state more or less closely. These patients are usually quiet and fairly obedient.

Physical signs, frequently overlooked by their minimal and isolated development, are present in the majority of the patients. Pupillary anomalies, modified knee-jerks, clonus, Babinski, nystagmus, ocular pareses, noises, mild ataxias, weakness of the bladder, mild changes in speech and in writing, are among the more frequent of these syphilitic signs. There is also marked increase in lymphocytes in the cerebrospinal fluid, and half of the cases show a positive blood Wassermann.

The psychosis may occur early, may be late after infection, and may even be a part of a congenital syphilis. The course is usually very chronic, and it may show long-standing remission after a fairly subacute course. The usual later development is that of peculiar dementia, occasionally accompanied by the negativisms, mannerisms, stereotypies, and other peculiarities known to occur in the more classical *præcox* situations.

Kraepelin is inclined to believe that such syphilitic psychoses are not infrequent. They are separated from dementia *præcox* at times with difficulty. Pathologically their syphilitic nature has been demonstrated, particularly in certain patients studied by Plaut and Alzheimer. (Plaut, *loc. cit.*)

Their affinity to certain tabetic psychoses is of much interest. The possibilities of syphilis, plus dementia *præcox* should always be borne in mind, but the discussion of this aspect of the problem must be found in works on psychiatry (Bleuler).

Tabetic Psychoses.—These can be outlined only categorically. One distinguishes:

- (a) Mild psychic disturbances (depression hypochondriasis, irritability, etc.).
- (b) Taboparetic association (cerebral syphilis).
- (c) Syphilitic dementia with tabes.
- (d) Associated psychoses with tabes.
- (e) Sensory syndromes—with paranoid coloring.
- (f) Tabetic psychoses properly speaking.

The most striking of this group are those with paranoid coloring.

Treatment.—The treatment of these syphilitic psychoses is that for cerebral syphilis in general and need not be repeated here.

8. SYPHILITIC MENINGOMYELITIS

Nonne has devoted a large portion of his noted monograph to a consideration of the lesions of syphilis of the spinal cord and its membranes. This is a general indication of its extreme frequency, yet most patients showing syphilitic lesions of the cord also show signs in the brain or its meninges. They are nearly all examples of cerebrospinal syphilis. For practical purposes, however, it has been found of value to arbitrarily divide this large conglomerate and discuss it under two

captions: Cerebral syphilis and spinal syphilis or meningomyelitis. This means simply that we are dealing with cerebrospinal syphilis with predominant cerebral and minor spinal symptoms on the one hand, and with predominant spinal and nerve root, with less prominent cerebral signs on the other. It has again seemed advisable to accentuate the purely arbitrary nature of all such classifications.

Principal Symptoms.—In considering meningomyelitis as a unit we find cause for further emphasis upon separable symptom groups. Within this conglomerate again practical neurology shows four fairly clear tendencies:

1. Syndromes due to pronounced meningeal implication.
2. Syndromes due to root and cauda equina disease. Radiculitis, neuritis.
3. Myelitis syndromes due to indiscriminate transverse disease.
4. Syndromes of less extensive transverse lesions and fiber tract isolation. System syndromes.

A combination of all would make a complete meningomyelitic syndrome. This is a not unusual picture in a rapidly developing case; in its more chronic course the emphasis seems to be laid upon one or another of the just mentioned groupings.

These are characteristic syndromes of the early and secondary stages of syphilis. The syndromes may develop within a few months after infection, or only come on after many years. In the former case the acute myelitic changes are frequent, also root lesions (many neuralgias, sciatica, etc.). The later developing cases show more the systemic lesions and gradually advancing meningopathies (later secondary meningitis), with compression (spastic) phenomena.

In all one expects to obtain a positive blood Wassermann; cerebrospinal fluid Wassermann is negative, save with large quantities of fluid; lymphocytosis is frequent—often the cell count being very high, always indicating the grade of meningeal involvement. The lymphocytes are not found before the stage of roseola; are abundant in the active secondary stages, and less frequent in the tertiary stages of a meningomyelitis. The protein content varies considerably.

1. **Meningeal Syndromes.**—Severe pains are signs of meningeal involvement. They shoot across the shoulder-blade, in the neck, across the hips, dart down the arms or legs and cause stiffness of the neck, the shoulders and the thighs. The spinal column is usually sensitive to pressure, and to percussion, and local intensities may show both these signs, and also the peripheral signs of a definite zone localization.

Pain is frequently preceded by paresthesiæ, the crawling of ants, numbness, and coldness. There is then a gradual increase in the reflex excitability of the cord, due to pressure and evidenced by increased knee-jerks, possible Babinski sign, Oppenheim or Chaddock signs. When pressure is exerted in the sacral segments, bladder and rectal disturbances are frequent.

With chronic meningeal thickening these pressure symptoms increase markedly, and spastic parietic phenomena augment, especially when

gummata add their special pressures. Gummata may give rise to a "cord tumor" syndrome.

Root Syndromes.—Here pain is frequent and neuritic atrophies appear. Sensory losses of a root distribution are in evidence. Many show the characteristic reversal of epicritic touch loss being less extensive than protopathic pain loss as pointed out by Head as pathognomonic of radicular lesions.

The atrophy of the muscles also follows the radicular distribution.

Many obstinate neuralgias are due to syphilitic radicular disease. Possibly one-half of the sciaticas are of this nature. Dejerine has put them as high as 80 per cent. in Paris. A very large proportion of the brachial neuralgias, so long looked upon as rheumatic or gouty, or what not, are due to a syphilitic root-meningitis.

Neuritic muscular atrophy, from pressure on the anterior roots, is further complicated by pressure on the anterior horns by the thickened meninges. Thus very anomalous atrophies result. When occurring in the seventh cervical and first dorsal region one obtains classical Klumpke paralysis with dilatation of the pupil, and narrowing of the palpebral fissure of the affected side. Lower localizations result in intercostal palsies, back muscle atrophies, hip girdle, thigh, or cauda lesions. In this latter situation striking dissociations are obtained, as in the upper arm region, and radicular sensory disturbances and lost reflexes are the rule with atrophies. (Plate XXXVII, Fig. 1.)

Myelitic Syndromes.—These indicate the complete involvement of the cord, and also point to intramedullary vascular disease, rather than to a meningeal lesions. Complete flaccid palsy is the usual lesion. This is combined with sensory loss as well. The completeness of the sensory loss varies considerably, and points to the severity of the lesion. Absence of a lymphocytosis points to a purely vascular and usually focal lesion within the cord. The bladder and rectal functions are implicated as well.

In the regressive stage an increase in spasticity marks the subsidence of the inflammatory reaction, and many anomalous syndrome mixtures result. This phase of meningomyelitis offers abundant opportunity for a very heterogeneous syndrome.

A Brown-Séquard complex, poliomyelitis, tabetic syndrome with atrophy, amyotrophic lateral sclerosis syndrome—these are but a few of the possible combinations.

Spastic System Syndrome.—These occur not so much as residuals of the previous myelitic changes, nor as due to meningeal compressions, but represent disease in or about the long motor tracts, tractus cortico spinalis especially. They give rise to the forms of primary lateral sclerosis (Erb), some combined scleroses, and particularly to clinical pictures closely resembling multiple sclerosis.

Special localization of one or more of these forms of meningeal syphilis give rise to the special forms of hypertrophic cervical pachymeningitis, which have been described by Joffroy and Charcot, and to Kahler's disease.

PLATE XXXVII

Fig. 1



Meningomyelitis. Radiculitis. Degeneration of Burdach's Columns.

Fig. 2



Brain of Juvenile Paretic showing Cortical Atrophy and Pial Attachment.

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In *hypertrophic cervical pachymeningitis* one finds an enormous thickening of the meninges, with or without gummata, and located in the cervical region. Here root and compression symptoms are present. Pains in the neck and shoulder, stiffness of the cervical spine, shooting pains down the arms. Sensory loss may then show, particularly to pin prick, with relatively intact sensibility to cotton-wool. The ulnar and median are particularly implicated. Fibrillary contraction of the muscles, atrophy and loss of electrical excitability occur. The preacher's hand is one of the frequent expressions of the involvement of the brachial plexus in the cervical meningitis.

Treatment of Spinal Syndromes.—The treatment of the various modifications of meningomyelitis, of radiculitis as syphilis, need not be considered further in this place. Enough has been said concerning the treatment of nervous syphilis as such to obviate repetition. The various syndromes mentioned on the preceding sections make up a very large quota in nervous syphilis. Syphilitic radiculitis (mostly sciatica, not infrequently brachial) is an extremely common syndrome in private as well as in hospital practice, and one which reacts very favorably to energetic specific therapy.

As in tabes, the Swift-Ellis procedure is logically applicable to other spinal syndromes. Here it is that one can expect to get most valuable results if direct application of a spirocheticidal remedy is to prove of any advantage at all. In the few patients thus far under treatment the results obtained have been hopeful. The table of one such patient is presented by courtesy of the writers (see page 412).

A further striking and rapid result obtained by the use of intraspinal injections alone is highly significant in a case of syphilitic spinal meningitis (radiculitis) (see page 413).

But this section will be devoted to the general care of the syphilitic patient, and the treatment of certain complications which may arise in the course of the various syndromes.

Diet is of some importance. Alcohol in all forms is to be avoided. Occasionally it is of value as a mild gastric irritant. Individual habits of diet may be uninterfered with if they have never shown particularly bad results. Dietary fads and superstitions should be avoided as far as possible. There still exist, even in modern works on dietetics, many of the legends and myths relative to foods which originated in their use in sacrificial and religious rituals. Red meats, pork, underground vegetables; etc., these have been taboo for centuries—solely because of certain mystical or religious rites, not on the grounds of rational dietetics.

The human mind is still so primitive that it is hard to get away from its superstitions relative to foods. Hence we say the matter of the kind of food is relatively unimportant. In many instances the form of the food becomes of moment. In comatose, paralyzed, dysphagic patients great care is needed during feeding to avoid choking or the production of inhalation pneumonia. Rectal feeding, or tube feeding, may thus become a necessity.

History of U. C. B., aged thirty-eight years; syphilitic myelitis; duration, sixteen months; primary unknown.

Date.	Blood.		Cerebrospinal fluid.				Treatment.		Clinical condition.
	Wassermann reaction.	Liver antigen.	Cells.	Noguchi globulin.	Wassermann reaction.		Intravenous.	Intraspinal.	
					Liver antigen.	Heart antigen.			
1912 Dec. 3	±		50	+	0.2 c.c. ++		Complete paralysis of both legs.
Dec. 5	±		0.6 gm. neosalvarsan		
Dec. 12	—		32	+	0.2 c.c. ++	0.9 gm. neosalvarsan	30 c.c. 40% serum	Marked improvement.
Dec. 19	—		10	+	0.4 c.c. ++	0.75 gm. neosalvarsan	30 c.c. 40% serum	
Dec. 26	—		0.75 gm. neosalvarsan		
1913 Jan. 9	+		5	±	0.4 c.c. ++	0.1 c.c. ++	0.9 gm. neosalvarsan	30 c.c. 40% serum	Steady improvement.
Jan. 22	+		3	±	0.5 c.c. ++	0.4 c.c. ++	0.75 gm. neosalvarsan	30 c.c. 40% serum	
Feb. 5	++		7	+	0.4 c.c. ++	0.2 c.c. ++	0.75 gm. neosalvarsan	30 c.c. 40% serum	Slow but steady improvement.
Feb. 19	++		3	±	0.3 c.c. ++	0.1 c.c. ++	0.75 gm. neosalvarsan	30 c.c. 40% serum	

History No. 565. V. H.,¹ aged forty-two years; tertiary syphilitic meningitis (radiculitis); syphilis, nine years ago.

Date.	Blood.	Cerebrospinal fluid.				Treatment.		Clinical condition.
		Cells.	Noguchi globulin.	Wassermann reaction.		Intravenous.	Intraspinous.	
				Liver antigen.	Heart antigen.			
1912 Apr. 5	• + ±	124	++	0.1 c.c. ++				
Apr. 10	64	++	0.2 c.c. ++	30 c.c. 40% serum	
Apr. 19	17	+	0.2 c.c. ++	30 c.c. 40% serum	
Apr. 28	30	+	0.2 c.c. ++	30 c.c. 40% serum	
May 7	23	+	0.2 c.c. ++	30 c.c. 40% serum	
Sept. 12	4	—	Negative				

¹ Report furnished by Neurological Institute.

Patients with cerebral signs should be kept in bed, in a quiet and darkened room, and when mildly delirious as few friends as possible should be permitted to see the patient, and only those with sense and tact who can comprehend and thus help the patient to manage his phantasy thinking.

In many of the patients with meningomyelitis certain complications are apt to develop. These are bed-sores, cystitis, incontinence. Bronchopneumonia is frequent. Good nursing is extremely important in the early periods while the antisyphilitic remedies are making their impress upon the spirochetes.

Bedridden patients should be placed upon specially constructed air- or water-beds when possible. Great care should be exercised in maintaining cleanliness. Wet and soiled sheets are responsible for skin infections; hence pads or special means of taking care of urine and feces are indicated.

The skin should be carefully gone over every day, the back, heels, and buttocks protected, carefully cleaned with antiseptics and alcohol, and dusted with antiseptic powder. In myelitis patients, sacral bed-sores are prevented only by the greatest vigilance. The conflict in syphilitic myelitis is usually won as soon as the spinal changes commence to react to the medication. If a bed-sore does occur, it should be treated surgically.

Cystitis is a frequent complication of meningomyelitis, as well as of other forms of cerebral syphilis—of paresis and of tabes. The greatest care must be taken to avoid sepsis in the bladder and its extension to the kidneys. Catheters must be used only when needed. Neglect to catheterize is a not infrequent oversight in treating dull or comatose patients.

Urinary decomposition is checked by antiseptics, benzoates, urotropine; at times the bladder is in need of irrigation, boric acid, hydrogen peroxide, or other genito-urinary antiseptic should be employed.

Special bladder treatment in tabes is often illusory, especially when it serves to hide the fact that the bladder disturbance is due to tabes. Treatment of the bladder should go on *pari passu* with the treatment of the tabes. The notion that cure of a tabes may come through the treatment of the bladder is superstition, if not worse.

The nervousness, irritability, restlessness, and insomnia of many of these patients in the beginning of their nervous syphilis should call for careful inquiry, and not the lazy handing over of bromides and hypnotics. It may be stated that the signs just enumerated should never be treated by bromides and hypnotics until the physician knows what he is treating. *Nervousness; irritability, sleeplessness as such, should never be treated.* Why is the patient nervous, irritable, sleepless? One is too prone to accept the lay idea that such signs may come into existence without cause. They have either a psychical or a bodily origin. If the former they should not be treated by drugs. Such is superstitious, magical therapy. If they are of somatic origin, this should be inquired into. The symptomatic therapy of the conditions just

mentioned leads to most appalling drug habits, and most dramatic tragedies. When too late one finds that "nervousness" had too real a disease process behind it.

The pains of meningomyelitis, often neuralgic, neuritic, may need special attention other than the specific medication. The analgesics already mentioned while speaking of the pains of tabes (p. 381) may be used, but never should they be employed if one simply is treating pain independent of its etiology.

9. CONGENITAL OR HEREDITARY SYPHILIS

It is thought that Paracelsus (1529) was the first to teach that syphilis might be transmitted to the child through the sperm cells. Ambroise Paré, a hundred years later, spoke of children coming from their mother's womb affected with this disease. Astruc, two hundred years later, made the definite statement. John Hunter (1786) introduced confusion into this, as in all of the rest of the syphilitic questions, by his arbitrary rejection of all of the evidence. Fournier in recent times has given the most extended studies which hardly exaggerate the entire situation. At all events congenital syphilis is not by any means rare, and presents itself in a vast variety of clinical forms.

Effect of Hereditary Syphilis.—Serological studies have thrown much light on the question of the mode of transmission. This cannot be entered upon here. These studies, as particularly carried out by Plaut, Mott, and others, have shown the enormous importance of transmitted syphilis in the pernicious effects upon the nervous system. Linser, moreover, has shown that two-thirds of the children of syphilitic parents show a positive Wassermann reaction, although much fewer show signs of congenital syphilis.

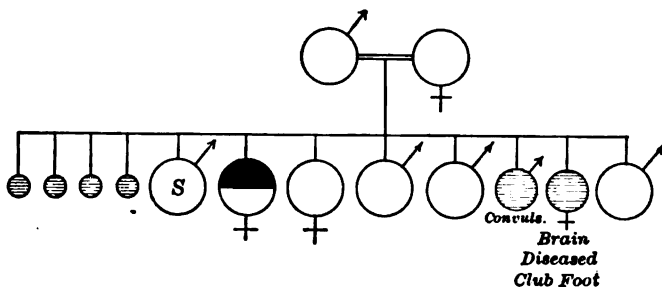
It may be recalled that Fournier stated the proportion as high as 98 per cent., and that 68.5 per cent. of the children died. This does not include the aborted offspring. Should these be reckoned one could obtain a true idea of the morbidity of syphilis in the young. Hochsinger reports an interesting group of cases in this connection. In 72 families there was paternal syphilis. The mothers were not syphilitic. Seventy mothers gave birth to 307 children—110 stillborn, 166 syphilitic, and 31 healthy. The healthy children were all the last born save in four instances.

Of the children of tabetics and paretics, one obtains the same story from Mott, Mendel, and others. Either no children, many abortions, many dead children, few living, and no one knows as yet the fate of these. Certainly one-half are doomed to disease and disorder of the nervous system.

It would appear that the common effect of such syphilitic infection is to reduce the resistances of the body and its powers for full development both in the general body and nervous tissues. Syphilis diminishes the vital energy of the germ plasm prior to conjugation, and

can cause pathological variations in nervous structures, just as it can transmit the disease through the germ cells. The abundant studies on alcohol and its influence on the germ cell affords an analogy in understanding how this takes place with another type of toxemia.

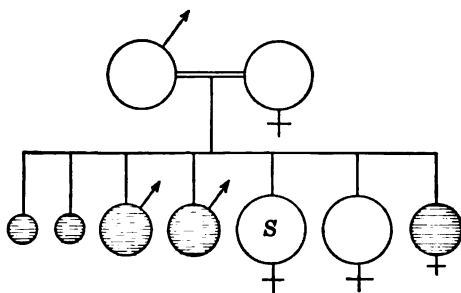
FIG. 31



Congenital syphilis: Mother syphilitic. Four children born dead; fifth, frail, skin syphilitic; sixth, paretic at fourteen years; seventh, well; eighth, well; ninth, well; tenth, convulsions at ten months; eleventh, brain disease and club-foot; twelfth, well. (Mott.)

The classical formula of Fournier seems to hold—abortion, dead child, early death, living healthy child. This is in need of amendment; it is worse. The formula reads. Complete sterility, miscarriage, abortion, stillbirths, children dying in infancy or convulsions, marasmus, meningitis, hydrocephalus. Then follow children who are comparatively healthy, but who in later life develop late hereditary syphilis.

FIG. 32



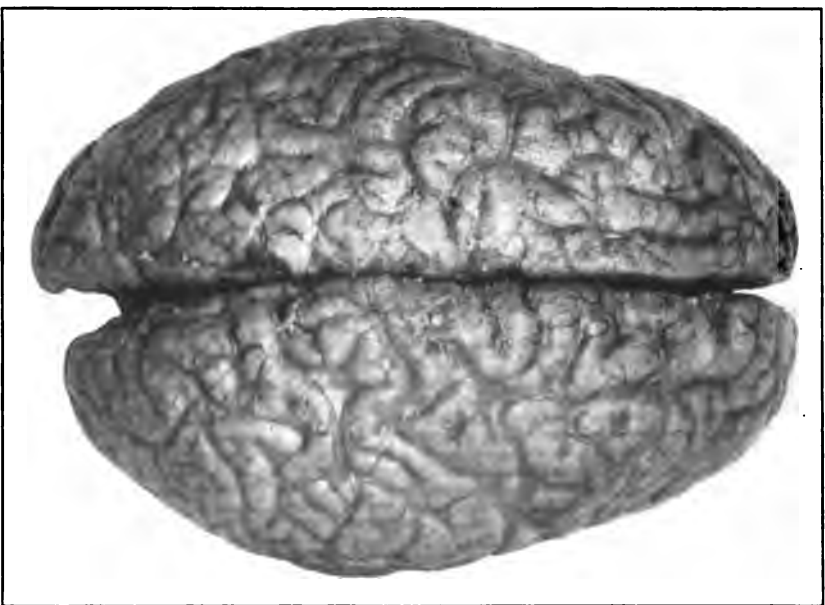
Congenital syphilis: Two miscarriages; two children dying at seven and fifteen months; fifth child dying at age of twenty-three of chronic syphilitic arteritis, small hemorrhagic softenings in basal ganglia; last child died at age of sixteen months. (Mott.)

Mott has given us a series of instructive family trees, which are worthy of reproduction.

The general problem, notwithstanding its interest, must not detain us here, nor can we insist upon the general signs of hereditary syphilis, save but to say that they tell but a small part of the truth. What information is obtainable about the nervous system? A late study

PLATE XXXVIII

Fig. 1



Brain of a Healthy Individual.

Fig. 2



Brain of a Congenital Syphilitic Idiotic Child.

U.S. 1

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by Hochsinger (1911) says that of 208 children of syphilitic parents who had been under observation over four years, 89 (or 43 per cent.) had some disease of the nervous system. Of these there were 9 cases of hydrocephalus, 2 of Little's syndrome, 6 epileptics, 2 paresis, 1 tabes, 6 Argyll-Robertson pupil, 36 extremely neurotic, 5 hysterical, 14 chronic headaches, 10 imbeciles. This is in strange contrast to the statements of Jonathan Hutchinson, who but a few years ago taught that hereditary nervous syphilis was negligible.

It has been abundantly shown that nearly every form of adult syphilis of the nervous system can be encountered in hereditary syphilis, and, as Mott well says, if congenital syphilis were not so fatal to infant life, the number of people suffering from brain disease, from syphilis, would be appalling. It would, therefore, be of little service to repeat what has already been written regarding nervous syphilis of adults as it appears in children, and the present discussion will be limited to a consideration of such forms of juvenile nervous syphilis as are present only in children. These are, particularly, certain forms of feeble-mindedness, of hydrocephalus, ependymitis, Friedreich's ataxia, primary optic atrophy, and encephalitides or encephalomalacias, leading to various hemiplegic syndromes, often loosely grouped together as Little's disease. Juvenile paresis and juvenile tabes are among the commonest congenital disorders of later infancy or adolescence.

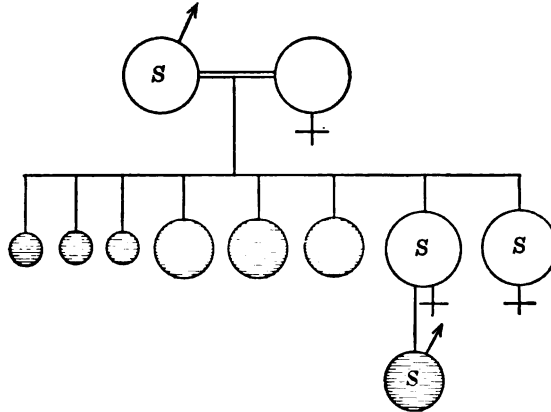
Congenital syphilis of the nervous system may show itself before or at birth; it may develop in the earliest infancy or in adolescence; it may develop as late as 20, or even 42 to 43 years (Müller). How long may the virus remain latent, finally to develop a definite syphilitic reaction? As yet the answer will depend upon the general bias of the answerer, rather than upon empirical data. It would appear from the evidence to hand that no definite age limit can be put upon the time when such a latent factor becomes activated by causes as yet unknown. That an activator of some sort plays a role in such disorders as tabes and paresis seems at present a justifiable hypothesis. When a newborn child known to have been syphilitic biologically can be followed throughout life, his serum reactions being tested from year to year, as is now being done, then the question can be finally decided.

In this connection a line may be devoted to the subject of congenital syphilis "unto the third generation." While of late years doubts have been accumulating relative to this matter, certain positive cases are being reported. The following chart illustrates an observation of Mott's recently reported (1912).

The *rationale* of this seems plain in view of the observations of recent years made by Levaditi, Bab, and others, that *Treponema pallidum* may be found in the ovum, and in an apparently resting stage similar to the resting stage known for other flagellate protozoa closely allied to the organism causing syphilis. Jonathan Hutchinson remains skeptical, but as many of the facts concerning which he has remained skeptical have been shown to be otherwise, notably juvenile tabes and paresis, it is probable that, like the opinions of John Hunter relative to syphilis,

ultraconservatism is a psychologically interesting individual phenomenon rather than a sound pragmatic instinct.

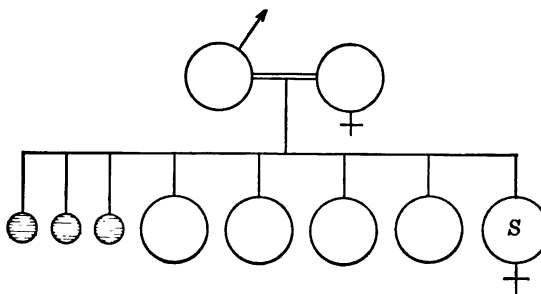
FIG. 33



Congenital syphilis "unto the third generation." Syphilitic father of first generation. 2d. Three premature births, two dead children, and one dying at sixteen months. Congenital syphilitic next, with Hutchinson teeth. This patient married, and had a child with snuffles, who died at age of six weeks. Remaining sister had infantile hemiplegia, Hutchinson teeth, keratitis. (Mott.)

The fact of the whole matter is that opinions and statistics relative to congenital syphilis of the nervous system, supported by clinical observation alone, and uncontrolled by the available biological tests,

FIG. 34



Congenital syphilis: Three miscarriages; then five children born alive and well. Last child snuffles, Hutchinson teeth. Did well in school, then deteriorated, noisy, maniacal; thought to be parietic. Autopsy showed generalised cerebrospinal gummatous meningitis, perivascularitis, and endarteritis.

are insufficient approximations, and very insecure. When relied upon for negative purposes they are harmful to the advance of thought, and detrimental to the relief of sick humanity.

Notwithstanding the importance, and often the strikingly gruesome character of these late appearing congenital cases, the attention of

the practitioner should be riveted upon the numerically preponderant miscarriages, stillbirths, and early syphilitic deaths if he would get in the right attitude toward the therapeutics of this disease, as it affects the nervous system. Some remarks upon the psychological control, the so-called moral attitude toward sexual freedom, as one of the burning factors in the present cultural struggle for more efficient adaptation have been made in those paragraphs dealing more directly with therapy of cerebral syphilis.

Here a return must be made to the clinical forms met with by the physician.

In congenital syphilis, as has been stated, one finds a replica of what has been found in adult syphilis. Pathologically speaking, the lesions are nearly always combined. There is a variable composite of end-arteritis, of leptomeningitis, of pachymeningitis, gummata, large and small, localized or infiltrating, gummatous neuritis, diffuse degenerative changes in the cells of the spinal cord, in the basal ganglia, or of the cortex. Thus the clinical pictures are apt to be conglomerate, and almost unanalyzable. Those more accentuated trends which permit a nosological term will be considered here.

Hydrocephalus.—As a result of congenital syphilis this condition has been suspected for two hundred years. Hasse in 1828, Cruveilhier in his atlas, Van Rosen in 1862, and Virchow reported definite examples of it. It arises in these congenital forms largely from syphilitic disease of the cerebrospinal fluid producing structures—choroid, ependyma, or from definite obstructive factors in the cerebral foramina, gummata, vascular swelling obstructing the iter, etc.

It is a not uncommon sequel of congenital syphilis, and is undoubtedly more frequent than is realized. In Hochsinger's series of 362 cases of congenital syphilis 34, or nearly 10 per cent., were of hydrocephalus. In his series, which affords a fairly average review of the situation, the hydrocephalus began three to eleven months after birth; sometimes it was fetal. In 11 cases there were no nervous symptoms, *i. e.*, up to the time of reporting. In the others restlessness, sleeplessness, chronic vomiting, convulsions, contractures, nystagmus, and feeble-mindedness were the objective phenomena.

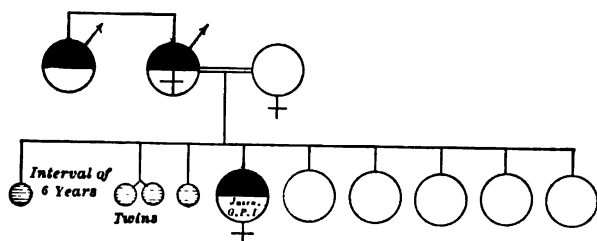
The more usual clinical picture is that of a child, boy or girl, from three to six months of age, who following, or not, an insignificant blow on the head, or some gastro-intestinal or bronchial disturbance, develops within a few days grave cerebral disturbances. There is great irritability and sleeplessness, screaming and kicking. The head is usually drawn back, the eyes and the fontanelles are apt to bulge somewhat. Vomiting is frequent, and there are signs of oculomotor involvement. Internal strabismus from paresis of the external rectus is not unusual. It is frequently preceded or accompanied by nystagmus and irregular pupils—often not responding to light.

Pain is present, as the child cries and struggles, and not infrequently the active movement of the arms—often highly spasmodic or convulsive—seem to try, in a blind reflex sort of way, to get at and brush

away the source of it, *i. e.*, the head, pulling the hair, grasping the head, rolling the head. Spasticity, rigidity, and other signs of intracranial pressure may at times be demonstrated. As a rule the temperature is only slightly or not at all raised, and the minor signs of an epidemic cerebrospinal meningitis, *i. e.*, herpes, temperature, flushed and spotted skin, are absent. The diagnosis of all of these infantile meningeal disturbances is fraught with much difficulty.

Feeble-mindedness.—Syphilis undoubtedly plays a much larger role in producing mental defectives than is suspected. The early statistics are comparatively worthless. They are quoted at great length even in modern works on idiocy, imbecility and the like. The English studies in particular—save the recent ones of Mott and his co-workers—only emphasize the league of the unconscious to suppress disagreeable truths which have unsuspected cultural values in the struggle against the repressions of the sexuality. We have already discussed at length this important topic, often lightly spoken of as Anglo-Saxon prudery, Puritanism, New England frigidity, in the paragraph on therapy of cerebral syphilis.

FIG. 35



Congenital syphilis: Juvenile paresis, at first considered as "imbecile." (Mott.)

The more correct appreciation of this chapter on syphilis and feeble-mindedness began with the studies of Fournier on parasyphilis. Those truths, somewhat uncontrolled, were forced upon him by his clinical observations. The early English, German, and American figures varied from 0.1 (Shuttleworth) to 17 per cent. (Ziehen). Whereas, the results following serological investigation start with the higher figures, and mount upward, in some cases as high as 40 per cent. The American figures available (Atwood and Clark) show that 20 per cent. of the idiots, imbeciles, and morons at Randall's Island, New York, were syphilitic.

We thus again see how groups become subject to analytical revision. Feeble-mindedness, so often regarded as a unicum, shows this tendency. Therapeutically one is called upon not to treat the name feeble-mindedness, but the reason behind it. As this paragraph does not deal with feeble-mindedness in its larger aspects (see chapter by Goddard, Vol. I) we shall limit ourselves to the purely syphilitic etiological subdivisions.

Intra-uterine feeble-mindedness is more or less a contradiction.

These children who would date their mental defect to disease going on in the uterus rarely live. Plaut expresses the opinion that feeble-mindedness may be regarded, so far as syphilis is concerned, as the result of an extra-uterine syphilitic disease undergone in infancy. In some there are signs of an acute brain disease. Some instances of hydrocephalus which recover show the signs of havoc in their inability to develop normally.

Many others show no acute stage, but fail to develop. Many recorded observations are available to show the very gradual development of mental defect, without convulsions or fever, altogether without signs of organic disease, which arrived at a definite termination, and which left behind as a result entirely stationary, perhaps even improvable, idiots or imbeciles.

That type of hereditary syphilitic child without any tangible disease, formulated by Fournier—his “*enfants arrières*”—who are described as unintelligent, simple, silly, limited children, always behind, this type not infrequently shows the Wassermann reaction.

Again, one is convinced by the researches of others that mental defect in less marked grade, or more properly speaking along more restricted or special lines, is allied with this broad group on the basis of congenital syphilis. Thus, Nonne reports cases of general irritable weakness of the nervous system. The patients are highly excitable, are extremely nervous, they are very moody, suffer from headaches, irregularities of appetite, sudden fits of passion—not associated with other forms of epileptiform analogies—and for whom mercury and the iodides worked wonders.

Still another chapter has been opened in this hereditary syphilis problem in its relation to mental defect. It concerns many so-called psychopathic children. These children are bright, but they show marked ethical defects. Here one can conceive of the mental defect in terms of limited cortical control to the effective response of the sexuality and to the nutritional instincts. These children want and arrive without going around by the circuitous routes devised by cultural standards. Anatomically one can posit a defect of certain cortico-cortical association areas on the basis of the syphilitic poisoning.

Taking the whole group of feeble-mindedness, it is evident from a reading of Dr. Goddard's chapter that clinically we cannot pick out the hereditary syphilitic child in all instances. Indeed, it should be emphasized that too much weight is given to the anomalies in physical structure—Hutchinson teeth, saddle nose, striæ about the mouth, prominent veins, scaphoid scapula, etc., if one rejects those who from the heredito-syphilitic class fail to show such anomalies. Nor can we recognize any certainly pathognomonic psychical anomalies. A careful neurological examination frequently aids in enlarging the group—particularly in the study of pupillary anomalies. The cytological tests are of the highest importance, and every child born of syphilitic father or mother should be systematically examined by these cytobiological methods.

Treatment.—In the presence of syphilis, as determined by the tests outlined, treatment by the methods already outlined will prove of immense value even in this apparently hopeless field. Every syphilitic child is entitled to the most that science has to offer.

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CHAPTER IX

THE USE OF SALVARSAN AND NEOSALVARSAN IN DISEASES OF THE NERVOUS SYSTEM¹

By HENRY J. NICHOLS, M.D.

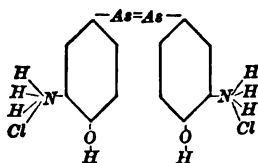
SALVARSAN

Introduction.—Salvarsan was elaborated as a means of killing spirochetæ *in vivo* and is the most efficient agent as yet known for this purpose. Its use in diseases of the nervous system is naturally indicated primarily in conditions which are due to invasion of the nervous tissues by spirochetæ. This invasion occurs chiefly in syphilis, and in active syphilis of the nervous system good results can be obtained, with proper precautions, by the use of salvarsan. Cases of acutal syphilis of the nervous system are, however, somewhat overshadowed by cases of parasyphilis—tabes and paresis—in which neither the etiology nor the results of treatment are as certain. Both diseases are believed to be due to a previous infection with *Spirochæta pallida*, but this organism has never been demonstrated in the lesions of either disease. On the other hand, the Wassermann reaction is positive in the blood in a considerable percentage of cases, and the serological findings of the spinal fluid also indicate an active process. In certain of these cases, especially of tabes, marked improvement in symptoms and in serological findings can be produced by salvarsan, but the results are not as constant as they are in conditions which we know are due to the actual presence of spirochetæ in the tissues. Aside from its specific action salvarsan has a tonic effect which can be used to advantage in certain other diseases of the nervous system, such as chorea, pellagra, etc.

Pharmacology and Methods of Administration—Salvarsan is a yellow powder which is made solely by the Farbwerke, formerly Lucius and Brüning, at Hoechst a/M, and is tested in Ehrlich's laboratory. It is sold in ampoules which contain 0.1, 0.2, 0.3, or 0.6 gm. As the substance oxidizes easily into a more poisonous compound, the tubes are exhausted of air or filled with a neutral gas and are hermetically sealed; hence no cracked or long opened tubes should be used.

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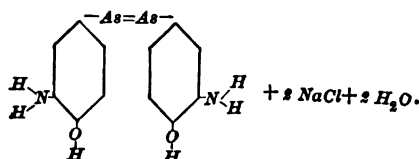
The powder, chemically, is di-chlor-di-amido-di-oxy arsenobenzol, and has the following formula:



In this substance the arsenic, which is the active portion, is attached to the benzol ring and to itself. In this way it is rendered inert until the combination is broken up and a large amount of arsenic can be safely introduced into the body. For example, as about one-third by weight of the substance is arsenic, in giving 0.6 gm. one gives nearly 3 grains of arsenic or the equivalent of nearly 4 grains of arsenious acid, but the usual toxic action of this large amount of arsenic is prevented by the chemical arrangement. When, however, the compound is broken up by combination with organic material, the arsenic, which is in a reduced trivalent form, acts vigorously in a nascent way and kills any protoplasm with which it comes into contact. Combination of salvarsan with spirochetæ seems to be effected by means of the amido and hydroxyl groups which seem to anchor the compound to the spirochetæ and thus bring the arsenic into action. A certain amount of the compound affects the tissues also, as is shown by the tonic action, but this amount is small, except in overdoses or in the presence of diseased tissues, which are more apt than normal tissues to anchor the compound.

As a general rule the actual administration of salvarsan should be put in the hands of someone who is versed in its use, whether or not he is a neurologist or a psychiatrist. To such an one the following directions will be superfluous, but they are included in order to assist anyone who may be starting in this work, and also to give the clinician an insight into the pros and cons of prospective treatment for his patient.

The powder itself is used in the form of a suspension in oil for intramuscular injection. It is preferable, however, when possible, to give the substance in a soluble form. The powder is easily soluble in hot water and forms a clear yellow solution, which has a decidedly acid reaction to litmus. This solution has been used for injection, but is usually regarded as too caustic on account of the acid radicles, and an alkaline solution is preferred. This is prepared by adding sodium hydrate. The first change on the addition of sodium hydrate is the formation of the neutral base as follows:



This base is insoluble in water and was used in the Weichselmann method of subcutaneous injection, but this form of administration has been almost entirely given up on account of the difficulty of absorption of the injected material. On further addition of alkali the base is redissolved as a sodium salt, which is alkaline to litmus. This is the soluble form in which the substance is given intramuscularly and intravenously.

The intravenous method of administration is unquestionably the method of choice, both as regards the comfort of the patient and the certainty of application of the preparation. Whenever possible, therefore, this method should be used. A certain amount of technique is, of course, necessary, and when it is not possible to carry out the procedure properly one should fall back on the intramuscular method of the alkaline solution or of an oily suspension. Each of these methods have advantages and disadvantages. The alkaline solution is soluble, but it is also quite painful. The suspension in oil is less painful and is easier to prepare, but it is in an undissolved form. Still it is probably the most useful form for intramuscular injection. The drawback to any intramuscular injection is the uncertainty of absorption; occasionally necrosis of the muscle occurs, with permanent encystment of some of the drug. When absorption is good the intramuscular injection produces a more lasting effect than the intravenous injection, but the same effect can be obtained by repeating the intravenous injection.

Injection of the Oily Suspension.—Articles required: sterile mortar and pestle; sterile liquid paraffin or iodipin; sterile dry syringe with good-sized needle; sterile castor oil. The desired amount of powder is shaken into the sterile mortar; about 1 c.c. of oil is added, and the powder is thoroughly mixed; enough more oil is gradually added with constant stirring to produce a fairly liquid mixture in 2 to 4 c.c. The mixture is drawn up in a sterile syringe which has been lubricated with a few drops of sterile castor oil. The patient lies on his stomach; the skin of one buttock is disinfected with tincture of iodine, which is washed off with alcohol after three or four minutes. The injection is given into the gluteal muscles in the middle third of a line from the anterior superior spine to the tuberosity of the ischium. The injected area should be well massaged and the patient should keep quiet for a day at least after the injection. The principal point in the technique is dryness of the syringe and needle, because salvarsan is hygroscopic, and if any water is present salvarsan takes it up and forms a sticky mass which interferes with injection. Some workers add to the oil a small amount of camphor and creosote for their antiseptic and analgesic properties.

Intramuscular Injection of Alkaline Solutions.—Required: sterile glass-stoppered bottle, 1 to 2 ounces, containing glass beads; test-tube of sterile water; 4 per cent. solution of sodium hydrate; 5 c.c. pipette; stirring rod. The tip of an ampoule is nicked with a file and struck off, and the powder is shaken into the sterile bottle. About 10 c.c. sterile water is boiled and poured on the powder; by stirring with a sterile glass rod the powder goes into clear solution. For each 0.1 gm. of the

powder, 0.7 to 0.8 c.c. of 4 per cent. NaOH is now added, or a corresponding smaller amount of a stronger solution. At first a precipitate of the neutral base is formed, and on shaking this gradually dissolves as a sodium salt. If a considerable amount of the base remains undissolved a few more drops of alkali should be added until only a few flakes remain undissolved. The resultant solution should be clear and yellow. If the solution is cloudy or blackish, this is due to some impurity in the water or sodium hydroxide and the solution should be discarded. For intramuscular injection the fluid is made up to about 20 c.c., and 10 c.c. are injected into each buttock. The patient should remain quiet one or two days.

Intravenous Method.—The fluid as prepared for the intramuscular injection is poured into about 250 c.c. sterile salt solution, which should be of temperature about 110° to 120°; the solution is then put into a gravity infusion apparatus, the tubing of which should already contain warm salt solution. If the veins of the forearm are prominent, one can be selected for injection without cutting upon it. The skin is sterilized; the vein is distended by a tourniquet placed around the upper arm; the skin over the vein is picked up and the needle first passed through the skin and then thrust into the vein. As soon as the needle is felt to be in the vein the tourniquet is released, and the clamp on the tubing loosened and the required amount is run in at the rate of about 40 c.c. a minute. If no vein is prominent, a tourniquet is applied above and cording is felt for and when located the vein is cut down upon and the needle inserted in plain view. The patient should keep at rest at least overnight.

The precautions necessary are: (1) The solution must be clear; a light cotton plug in the bottom of the infusion apparatus serves to take out particles of cotton, etc.; (2) the solution must not be over alkaline or thrombosis may result; (3) the salt solution must be made with *freshly distilled* water—old distilled water is apt to contain many organisms and give rise to severe reactions; (4) blood should not be allowed to flow back into the infusion tube; (5) the needle must be large enough to permit a good flow; (6) none of the solution should be allowed to escape into the tissues about the vein, as it will set up an irritation.

The reaction to an intravenous injection varies with conditions. In early cases, with best technique, there may be some fever which is due to the disintegration of large numbers of spirochetæ. Later in the disease, fever should not be marked if the proper technique has been followed, especially in regard to the use of freshly distilled water. Many of the violent reactions on record have been due to dead micro-organisms in the fluids used instead of to salvarsan. The symptoms of poisoning with salvarsan are, in general, those of arsenic, and consist in irritation of the stomach and intestines, in a scarlatiniform rash, and in parenchymatous degeneration of the organs, especially the liver, kidneys, and heart. There is some evidence that toxic symptoms are more apt to occur if a number of injections have been given already.

General Principles of Salvarsan Therapy.—In the course of two years certain general principles have been developed in regard to the use of salvarsan in syphilis which have a bearing on its use in syphilis of the nervous system. It is now recognized that salvarsan kills the accessible spirochetæ more quickly and more certainly than mercury; hence it may be of special value in producing quick results when such delicate and important tissues as the nervous tissues are involved. The most direct way to reach spirochetæ in the tissues is, of course, through the circulation, and this is accomplished by the intravenous method. It has also been established that in the great majority of cases all the spirochetæ are not accessible to the circulation, and, hence, if no further treatment is given the spirochetæ which have escaped the action of salvarsan will multiply and a relapse will occur. (Neuro-recidive, see Chapter VIII.) If the patient is in the secondary stage, these relapses may affect the nervous system, especially the optic, auditory, and the facial nerves. To prevent these relapses it is necessary to repeat salvarsan or to give mercury in combination with it, in the form of injections or inunctions.

Dangers.—The dangers of the use of salvarsan in syphilis of the nervous system are chiefly two; first the possibility of producing an internal Herxheimer reaction, and second the danger of the decomposition of the drug by organic lesions. The Herxheimer reaction was originally noted in skin lesions; it consists in a temporary accentuation of a lesion after treatment with mercury or salvarsan, and is supposed to be due to the liberation of toxins on the death of the organisms. For example, after salvarsan treatment a papule becomes brighter and more prominent and then disappears. The same process may occur in internal structures, and if it occurs in a nerve or in the brain, the swelling may produce serious results before resolution can occur, as, for example, when the phrenic or vagus nerve or structures in the internal capsule are affected. The second danger consists in the possibility of the drug being taken up and decomposed by old organic lesions, and again when this occurs in "the master tissues" the results may be disastrous.

Both the dangers may be avoided to a certain extent by the use of several small doses instead of one large dose; and while due caution should be used, in many cases it is justifiable to run certain risks because many cases are not influenced by mercury, and the only alternative to the use of salvarsan is permanent injury, the insane hospital, or death.

Salvarsan in Syphilis of the Nervous System.—The evidences of early involvement of the nervous system, while not so striking and important as those of later involvement, are very definite and are better appreciated now that we know their cause. When the spirochetæ become generalized they invade the lymph spaces, especially in the walls of the vessels, in the brain, cord, and nerves as well as in the skin; they have been demonstrated in these areas by sections. Here as elsewhere they lead to an accumulation of round cells which is supposed to be more marked

in cases with a papular eruption. These accumulations of cells exert pressure effects and these, with toxic influences, are manifested by changes in sensation, motion, and even of consciousness. The most common early symptom is, of course, headache; then comes paralysis of the facial or oculomotor nerves, then retinitis and affections of the auditory nerve, then convulsions. All these symptoms can be traced directly to the presence of spirochetæ in the meninges or walls of the bloodvessels, and can be directly influenced by salvarsan. Headache disappears almost by magic—paralysis of one side of the face is quickly replaced by tone and so on. In these early cases there is less to fear from anchoring of the substance by diseased tissue, and the Herxheimer reaction is not so dangerous, and doses of 0.3 to 0.4 intravenous, or 0.4 to 0.5 intramuscular, can safely be given. The effects are often brilliant; the immediate results, however, must be followed up by further combination treatment if relapses are to be avoided.

Later in the course of the infection the changes in the meninges and vessels are more profound and results are more variable. Such symptoms as disorders of speech, ataxia, altered pupillary and patellar reactions can be relieved, but there can, of course, be no restoration of destroyed tissue. There is more danger of decomposition of the preparation, but as was said above the alternative is such that in many cases the drug should be tried in small doses of 0.1 to 0.3 gm. and repeated after a short interval. The literature of the subject contains records of happy results in some cases, no improvement in others, and of unfortunate results, even death in others. Each case must be considered by itself, and whenever possible the results of serum examinations should be considered. If the symptoms seem to be due to an active exudative process the combined treatment should be cautiously undertaken. If the disability is due to an old lesion little result is to be expected, and more harm than good may be done by using salvarsan.

Tabes and Paresis.—The treatment of tabes and paresis has been so discouraging that on the introduction of salvarsan great hopes were raised in regard to the treatment of these diseases. These hopes were based partly on Alt's early experiences. He first used the drug on paretics and was able to influence the Wassermann reaction. It seemed that there must be some clinical equivalent of this change in the serum and a great many cases of each disease have been treated. The results, according to different authors, have varied from *nil* to something quite encouraging. Some workers consider these diseases as contraindications to the use of salvarsan, but such a view is certainly extreme.

Before discussing the results of treatment, it will be well to review briefly what we know about the etiology of these diseases. We may start with the generally accepted proposition that syphilis is a *sine qua non* for tabes and paresis. (Also see Chapter VIII.) But what is the exact relation? One of the best authorities, Mott, holds that these diseases are residual paralyses, that the nerve cells are exhausted by a previous infection, and as they cannot be replaced, they degenerate and produce effects long after the virus has left the body. This

explanation, as Mott says, is not altogether satisfactory. Some cases seem to fit in with this theory, but a majority of them do not. How, on this theory, can we explain the positive Wassermann in the blood and spinal fluid, the increased cell count of the spinal fluid, the increased globulin content, the accumulation of round cells about the vessels and in the meninges, and the progressive character of paresis? All these findings point to an active process rather than to a residual process. How can we explain the improvement some cases of tabes show under mercury and the iodides? If this theory be correct, what occasion is there for using salvarsan? As has been said, spirochetæ have never been demonstrated in the lesions of either disease, but it certainly seems that some virus is at work, and in some cases the virus behaves like a spirochete.¹ With salvarsan we can remove the symptoms and change the serological findings to normal. In other cases the results do not encourage the belief that a spirochete is the cause of the lesions. It is, of course, possible that in these cases we are dealing with a different strain of the organism or with resistant forms, but for the present we must admit our ignorance of a satisfactory explanation.

Tabes.—Coming now to actual cases of tabes, we find that they fall into two classes as judged by the presence or absence of evidences of an active process—such as the Wassermann reaction in the blood or spinal fluid, the presence of a hyperlymphocytosis, etc. (See discussion of clinical types in Chapter VIII.) If one has to lay down a general rule, it would be that salvarsan is indicated only in the positive class of patients, but the rule would have a good many exceptions. Some positive cases would not respond, while a few negative cases would show a great improvement. Still there is a good deal of evidence to support the rule. For example, I have seen a skilled decorator whose hands were becoming so ataxic that he could no longer work. He had had syphilis seven years before, and had had three years of mercury by mouth; his Wassermann reaction was +. He was examined by several competent clinicians and pronounced an early tabetic. He was given an intramuscular injection of 0.5 gm. salvarsan in alkaline solution; in three weeks his reaction became negative. At the same time his ataxia disappeared and he returned to work. His reaction remained negative for several months and finally returned, without a recurrence of symptoms, in the eighth month. This case behaved exactly like a case of spirochetosis, and a number of cases of the same kind have been reported. The same results could probably have been obtained, in a longer time and with more discomfort, with mercury. In this class of cases salvarsan should be given a trial with the understanding that it is a trial. There is not much danger of a Herxheimer reaction, and if the patient is in good health otherwise, doses of 0.3 to 0.4 gm. can be given intravenously, or 0.4 to 0.5 gm. intramuscularly. If the patient is debilitated, smaller doses should be given and repeated, and if

¹While these pages were going through the press there came the demonstration of spirochetæ in the brain of paresis.—Ed.

improvement occurs mercury should be given to prevent a relapse. In the cases that show no evidence of an exudative process little is to be expected from salvarsan, except in temporarily relieving pain. Surprisingly good results occasionally occur, but cannot be rationally explained.

Paresis.—The results in the use of salvarsan in paresis have been disappointing. (See Chapter VIII.) It is true that all the evidences of an active process are present, and that the Wassermann reaction and cell count can be occasionally reduced; clinical improvement, too, occurs; but, in the opinion of most alienists, this is in the nature of a remission more than as a result of treatment, and the prognosis is not changed. In well-advanced cases the administration of salvarsan, like any other heroic treatment, seems to do more harm than good, and hastens the end. In early cases, especially those in which cerebral syphilis seems to grade into paresis, there seems to be some hope of arresting the process. As in the case in advanced cases of cerebral syphilis, the dangers of toxic action are considerable, and small doses of 0.1 to 0.3 gm. should be used.

Salvarsan in Other Diseases.—Salvarsan has been tried on a great variety of other diseases of the nervous system and seems to have no specific action, but acts simply as arsenic. The argument for its use sometimes rests on the large amount of arsenic that can be introduced, but, as far as we know, when the compound does act, it acts simply as a result of partial decomposition and the undecomposed portion does not act. Hence the only advantage it seems to have over arsenious acid or its salts is in the certainty of application by the intravenous route.

NEOSALVARSAN

The possibilities of chemotherapy are illustrated by the fact that since this chapter was written a new form of Ehrlich's spirocheticide has become available for the profession—number "914," or neosalvarsan. The chemical action of this preparation is essentially the same as that of salvarsan, but it has two advantages in practice: it is directly soluble in water, requiring no preliminary treatment, and it forms a neutral solution. Ehrlich succeeded in making these improvements by replacing one of the H atoms of one of the NH_2 groups by formaldehyde sodium sulfoxylate $\text{CH}_2(\text{O H})\text{OSONa}$.

Neosalvarsan was first sent to Schreiber, of Magdeburg, for trial, and he used it intensively in intravenous form, giving one injection every other day for four doses, and also advocated the intramuscular injection as much less painful than the old form. In a short time, however, it became evident that the body could not tolerate such frequent doses, as complications occurred in the form of peripheral neuritis which was clearly due to the drug itself. Schreiber, therefore, advised that the interval between injections be lengthened. Some authors have claimed that neosalvarsan is more toxic and less effective than

salvarsan, but the consensus of opinion is that it acts equally as well and is easier to give.

Neosalvarsan arrived in this country in April, 1912, and I have given it a thorough trial and feel that in time it will entirely replace the older form. It is a yellow powder slightly darker than salvarsan and is one-half heavier, *i. e.*, 0.9 gm. of neosalvarsan is the equivalent of 0.6 gm. of salvarsan. Rabbits tolerate fully twice the weight of salvarsan, as the fatal dose, 0.2 gm. per kilo, intravenously, instead of 0.1 gm. Its action on spirochetæ in chancres of rabbits in proportionate dose is fully as good as that of salvarsan, as they disappear within twenty-four hours. The action of neosalvarsan on skin lesions and also on the Wassermann reaction has seemed to me to be fully as good as that of salvarsan. Its effect in cerebrospinal syphilis will be discussed directly.

I cannot recommend the intramuscular injection of neosalvarsan as possessing any great advantages over that of salvarsan. Swift has shown that in rabbits the necrosis of tissue is not as great and that the absorption is more rapid, but its administration is decidedly painful in any form, and my patients have protested vigorously against a second dose. As was said above, the equivalent of an intramuscular injection can be obtained by repeating intravenous injections.

The technique of an intravenous injection is like that for salvarsan, except that the powder is dissolved in about 200 c.c. of properly prepared 0.4 per cent NaCl solution and is given *immediately*, at *room temperature*. The intramuscular injection is prepared by dissolving the powder in 20 to 30 c.c. freshly distilled water and injecting one-half in each buttock. The doses are proportionate to those of salvarsan, 0.9 gm. being the maximum for an adult.

Neosalvarsan can produce excellent results in cerebrospinal syphilis, as is seen from the abstract of the following cases: C. G. F., aged thirty-two years; fireman; no history of syphilis. In June, 1912, began to have severe headaches. July 17 consulted his physician with severe headache and temperature of 101°. July 21 became entirely confused. July 22 Wassermann reaction ++; Sp. Fl. Noguchi butyric acid test +; cells 90, Wassermann in fluid +. July 29 condition the same, with twitching of muscles; patient was given 0.6 neosalvarsan intravenously and inunctions of mercury and KI. In forty-eight hours the temperature suddenly dropped and remained normal; patient still confused. August 2 a second intravenous injection was given and in a few days the patient became rational. August 8 a third injection was given. Wassermann in blood and fluid negative, cells 31. Butyric acid test + -. The patient soon went on a furlough and in November returned to duty weighing more than ever, and in excellent general condition. Wassermann, December 20 —. This patient was headed for chronic dementia in an insane asylum and was saved by the Wassermann test and neosalvarsan. Full doses were used because there were no localizing symptoms as a contraindication and radical action was necessary.

A more striking case was one of nervous relapse observed by

Dr. Hough, of the Government Hospital for the Insane, and myself. This patient had been given two intravenous injections in the florid secondary stage and became apparently perfectly well. Three months later he developed a distinct psychosis, with paresis of the right side and motor aphasia. The cell count of his spinal fluid was 840 and we were able to demonstrate *Spirochæta pallida* in his fluid by inoculation into a rabbit. Dr. Hough gave the patient three injections of neosalvarsan, two intravenous and one intramuscular; his cell count dropped rapidly to normal and his symptoms disappeared so completely that in three months he was discharged, practically well.

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CHAPTER X

THE TREATMENT OF CEREBRAL HEMORRHAGE, EMBOLISM, AND THROMBOSIS

By FREDERICK TILNEY, M.D.

CEREBRAL HEMORRHAGE

It has been the custom to regard cerebral hemorrhage, embolism, and thrombosis as diseases of the brain. Symptomatically and pathologically there is much to justify such a view; yet, from the broader clinical standpoint, this attitude toward the question cannot fail to be prejudicial to the most efficient prophylactic and preventive treatment for these conditions.

Cerebral hemorrhage, of other than traumatic origin, should be looked upon as a local expression of a systemic pathological process. It is but one of the many possible manifestations which indicate a failing resiliency on the part of the tissues in general and the blood-vessels in particular. In the greater number of cases this is also true of cerebral embolism and thrombosis. These latter conditions, as a rule, have an antecedent history, the interpretation of whose cardinal features points to an extensive involvement of the blood-vascular apparatus. The fact that cerebral hemorrhage and infarction cause such pronounced symptoms should not be allowed to draw the attention away from the underlying causes which go to make up the pathological history of these diseases. If, therefore, cerebral hemorrhage, embolism, and thrombosis be regarded as manifestations of systemic diseases—and there is every reason that they should be so regarded—it is then possible to recognize certain periods or phases in the course of their development with reference to their clinical behavior. This statement, while of general application, is subject to certain exceptions, as will appear later in considering some types of cerebral embolism.

Periods.—The first of these periods referred to is the Period of Preparation. It represents a phase of the organism during which the katabolic process is dominant and the vascular system is undergoing retrograde changes as a result of more or less obscure influences.

The second period is the Period of Insult, in which some part of the vascular system in the brain either ruptures or becomes occluded.

The third period is the Period of Paralysis, in which, as a result of the sudden vascular changes in the brain tissue, certain symptoms make their appearance, due to the interception or abrogation of definite cerebral functions.

Each of these periods has a pathology of its own. It is doubtless the case that each period has its own etiology as well, and hence will demand special treatment.

The Nature and Mechanism.—Not only do the severity and definite character of the symptoms following cerebral hemorrhage make it appear that this type of hemorrhage occurs more often than in other organs, but actual computation shows that the spontaneous rupture of vessels in the brain is more frequent than elsewhere. The reasons for this may be found in the fact that brain tissue itself affords but little support to the arteries. In addition to this, these arteries do not anastomose and the arterioles are imperfectly covered by a muscular coat. The cerebral arteries have, therefore, but a scant margin of safety upon which to depend during periods of increased vascular tension and stress.

Types and Etiology.—Hemorrhage into the brain may be of two types, *i. e.*, capillary or spontaneous. Capillary hemorrhages occur as complications of acute infectious diseases, as a result of congestive hyperemia, acute encephalitis, or the congestion occasioned by the pressure of tumors and exudates.

Spontaneous Hemorrhages.—Spontaneous hemorrhages are of much greater importance. They are caused by the rupture of one of the cerebral arteries and give rise to the symptoms usually described under the term of apoplexy. The rupture of such an artery is predetermined by a diseased condition of its walls, which thus weakened, present a series of miliary aneurysms. The degeneration in the vessel may be due to inflammation, calcification or sclerosis; it may possibly occur in the arteries of the brain only or, as is usually the case, it may be a more generalized process affecting the entire arterial system. Whether or not we are to call this condition atherosclerosis or arteriosclerosis makes little difference if we recognize that the essential change is a lessened resistance in the vessel walls. This change, moreover, has been wrought in the course of a gradual process whose direct causes are to be found in the increased strain sustained by the arterial wall as a result of heightened blood pressure, long continued or occurring at irregular intervals. In addition to this, the wall of the vessels may be inherently weak due to congenital defectiveness or disease; this furnishes another direct cause of arteriosclerosis.

If the hemal vascular system were concerned merely with the conveyance of blood from one part of the body to another, its task would be a relatively simple one and its liability to disease but slight. It bears, however, the greater responsibility of vascular adjustment. It determines the distribution of the blood according to the functional necessities of the various organs. It economizes in one region so that it may give a fuller amount to another region in actual need of increased supply, in this way it maintains a balance between the various organs according to their varying needs. The most important elements in this mechanism of balance are the arterioles. The small and medium-sized arteries are also active in this mechanism, so that when one part

is receiving an unusual volume of blood, its vessels dilate, while the arteries and arterioles in other parts become correspondingly contracted to the end that the normal average blood pressure of 120 mm. Hg. is maintained.

Although it is clear that the vascular system has a profound influence upon the activities of the entire economy, it must be quite as obvious that from the very intimacy of its relations with the several organs it becomes subject to influence from them as well. Intrinsic diseases of the digestive system or its adnexa, of the urinary, respiratory, or hemopoietic systems of necessity require the establishment of a new balance which may be to the ultimate disadvantage of the arterial walls because of an increased blood pressure. Such disorders as these in the organs of other than the vascular system may furnish the basis which determines a permanently elevated blood pressure and so subject the arteries to a deleterious strain. From their office as conveyers of the blood stream the vessels, particularly the arteries, lay themselves open to injurious influence from another source. Passing as they do to all parts of the body, they must encounter various products of metabolism; their tolerance to the greater number of these products is remarkable; but under abnormal conditions unusual products are met with and these, either by direct stimulation of the vessel walls or by excitation of the vasomotor centres in the nervous system, cause a contraction of the arteries with an attendant elevation of blood pressure. There is much evidence that intermittent and even prolonged arterial spasms due to these causes are of much more common occurrence than is generally thought to be the case. Such conditions are seen in intermittent claudication, Raynaud's disease, the vascular crises of the splanchnic area described by Pal and according to the recent studies of Lauder Brunton, the common forms of migraine usually called "sick headache." Increased blood pressure is undoubtedly an indication of injurious influences at work on the vascular system, and it probably is true that of itself it adds further to the damage being done.

Were it possible to sum up the causes which lead to increased blood pressure, it would be necessary to review all of the processes of metabolism giving rise to endogenous toxins, and all exogenous toxins, as well as the diseases peculiar to the several systems of organs and finally to recognize the inherent tendency of the arteries to lose their resiliency with advancing years. At present we have only a vague idea of these conditions and their possibilities. Yet we do appreciate that certain errors in diet, hygiene, and the general habits of life contribute in a marked degree to the vascular changes responsible for so much disease in the body. It is rarely the case that a patient comes under observation until the arteriosclerotic changes have gained considerable headway. In some instances, however, the vessels may present surprisingly little sclerosis and the patient yet suffer from the effects of high arterial tension due to arterial spasm. Such cases demand special care and study. Somewhere in the economy the process of metabolism is at fault; this fault if allowed to continue will inevitably lead to vascular

degeneration. In either event, in the case of clearly established arteriosclerosis or in the case presenting arterial spasm alone, the patient must be looked upon as favorable subject for a cerebral hemorrhage. This should be one of the chief possibilities against which the treatment shall be directed.

Diagnosis.—Cases of these two varieties definitely belong to the period of preparation for cerebral hemorrhage. With the recognition of a high arterial pressure (180 mm. to 220 mm. and above) the first question to determine is whether this pressure is due to arteriosclerosis or arterial spasm. Much assistance will be afforded in this inquiry if a careful daily blood pressure curve is kept for a number of days. A sustained high pressure without marked fluctuations would indicate a sclerotic rather than a spasmodic condition in the arteries. Certain precautions must be taken in obtaining the pressure curve, lest physiological variations be mistaken for pathological changes. If taken once a day, the reading should be made at the same hour each time and at a stated interval after a meal; the patient should always be in the same position and under about the same surroundings. Exciting influences should be excluded as far as possible and the patient's mental state noted at each reading, especially as to worry, depression and the like. Further, to secure accuracy in the pressure curve it is desirable that the patient remain quietly in the recumbent or sitting posture for fifteen minutes before the reading is taken. Fluctuations of 15 to 20 mm. on different days should not be regarded as contradictory to a sustained increased blood pressure. It is only when the curve shows marked depressions approximating the normal line with sudden, high elevations that the question of arterial spasm should be considered.

Treatment of the Period of Preparation—With the fact of a sustained high arterial pressure once established it becomes necessary to determine which of the organs may be particularly at fault. The kidney usually suggests itself first, and while its condition should in all cases be carefully investigated, important data will be overlooked unless other organs are also examined. This is especially true of the liver, which is apt to be enlarged or cirrhotic. The condition of the gastrointestinal tract may also reveal important factors contributing to the disease, and finally the heart and lungs.

In cases of arterial spasm presenting intermittent claudication, local pallors and hyperkinesis, splanchnic crises, and periodic attacks of migraine attended with high arterial pressure, particular care should be taken to discover, if possible, some toxic irritant sufficient to produce the conditions. In the main, the nature of such toxemias is still obscure. Interesting and suggestive in this connection, however, is the relation of anaphylaxis to arterial spasm. It has been conclusively proved by Schultze and Jordan that the anaphylactic symptoms in guinea-pigs which produce death in apnea are due to an obliterating spasm of the pulmonary artery and a contraction of the smooth muscles of the bronchioles.

Young and robust individuals often suffer from severe periodic

migraine. For several days before the migraine occurs the blood pressure will rise to 200 mm. Hg. or above. After the acute attack, the pressure diminishes to 140 or 150 mm. Hg. Headaches occurring on the average of twice a week subject these patients to dangerously high blood pressure the major part of the time. On a protein-free diet these cases often go for long periods without headaches, and even a slight amount of meat will frequently cause a violent attack of migraine. The possibility of the patient's sensitization to proteins should be considered in every case of spasmodic, remittent high blood pressure and a protein-free diet rigorously enforced for at least one week in order to make accurate observations. If this diet holds the increase of blood pressure in abeyance, a small amount of some specified meat may be given in order to test experimentally the anaphylactic tendencies of the patient to this particular protein. Following this, a rise in blood pressure indicates that the patient will do better without this protein unless an immunity can be produced.

Other toxins may occasion increase of blood pressure, especially those arising as by-products of enteric digestion such as indol and skatol. These substances are generally rendered innocuous by the liver and, in part, by the muscles. Their production is best controlled by limiting the amount of ingested meat, increasing the amount of outdoor exercise, free and regular catharsis, together with the use of the lactic acid producing bacteria, preferably in some of the artificially soured milks. The management of cases having high blood pressure due to intermittent arterial spasm is difficult in many ways. As a rule, the patient's general health is good and it requires much emphasis to impress upon him the necessity of carefully regulating his life and habits. Sedentary occupation should be relieved by recreation and outdoor exercise; overeating should be avoided and the use of alcoholic beverages interdicted.

In cases of high pressure due to spasm of the vessels, the pulse will gradually decrease in volume as the manometer pressure increases. Immediately before it becomes obliterated, however, the pulse will show several pulsations of nearly full volume. This has been called "ballooning of the pulse before disappearance." It does not occur in elevated blood pressure due to arteriosclerosis.

When the blood pressure is permanently high in the presence of arteriosclerotic changes, the problem of the treatment becomes more complicated. The patient is now actually in a condition favorable to cerebral hemorrhage, and this accident must not be regarded as among the least likely outcomes of his condition. The literature concerning the treatment of arteriosclerosis is one of the most extensive chapters of internal medicine, and too wide a subject to permit of more than a summary consideration here. It is extremely doubtful whether any therapeutic agents can repair the damage already inflicted upon the arteries. The problem of the treatment centres about the attempt to restrict the further advance of the disease and to instruct the patient to live within the limits imposed upon him by his sclerotic vessels. The

condition of the kidney, liver, and intestines should be carefully watched with special attention to the possible occurrence of any of the toxemias already mentioned. The drug almost universally recommended is potassium iodide, or else the iodine radical in some other form, as, for example, dilute hydriodic acid. In some quarters this medication has fallen into disrepute and is regarded as a mere fetish. Absolute proof against this contention is not forthcoming, but the approval of the use of potassium iodide is so general as to justify its application to all cases.

Diet.—In the matter of diet for these patients, it is difficult to make general recommendations which will be specific enough for particular cases. The application of dietetics is unfortunately limited by the fact that the digestive processes differ in different individuals, and the diet which may be beneficial for one person may be quite the reverse for another. This doubtless explains the great differences of opinion among dieticians in recommending the kinds of food best suited to any given disease. In this light the best advice as to diet is the injunction to be moderate in eating and to select such foods for a diet as have previously been proved to agree with the patient. To this must be added the general precaution of excluding the proteins or carbohydrates as the case may demand. Alcoholic beverages should be prohibited, for in spite of the fact that their use in moderation has been recommended, the interpretation of the meaning of moderation must not be left in the patient's hands. It is seldom possible for a patient to estimate this limitation correctly, or having made a correct estimate to live up to it, so that all things considered it is better to interdict the use of alcohol altogether.

General Hygiene.—The matter of general hygiene is of equal importance. The occupation of the patient should be regulated insofar as possible. The participation in unusually active business affairs, entailing great responsibility and anxiety, is distinctly disadvantageous. Sedentary habits should be avoided and a moderate amount of daily outdoor exercise recommended. Bearing upon the amount and kind of exercise in which patients suffering from high arterial pressure should be allowed to indulge, there has been much discussion. Jones' figures bring some interesting facts to light in this regard. They seem to show that whereas the exertion of such natural acts as laughing, defecation, coitus, and the like have been held responsible for the production of cerebral hemorrhage, in only a very few instances have these acts been in any sense causative. In 114 cases of cerebral hemorrhage, 18.4 per cent. occurred while the patients were in bed and 9.9 per cent. during sleep. "From this it appears that rest in bed and especially sleep protects, to some extent, against cerebral hemorrhage; yet a miliary aneurysm that is going to rupture rarely needs the aid of severe exertion for this consummation." These facts seem to justify the recommendation of a certain amount of outdoor exercise for patients suffering from permanently elevated blood pressure.

It is unfortunately the case that the majority of hemiplegics pass through the period which is preparing them for their apoplexy without

any knowledge of the ominous changes which have been occurring. Often cerebral hemorrhage is the first indication of arteriosclerosis or chronic nephritis. For this reason the remarks on the treatment of the period of preparation have perhaps more theoretical than practical value. They may, however, serve to call attention to the fact that there is such a period in the greater proportion of cerebral hemorrhages, and that all patients suffering from sustained high blood pressure with arteriosclerosis are potentially hemiplegics.

Treatment of the Period of Insult.—The Immediate Management of the Case.—A patient found in coma demands the closest investigation possible. The first fact to be determined is the presence or absence of hemiplegia. If the coma is not too deep this question may be readily answered by examining the muscular resistance of the legs and arms of the two sides of the body. The coma is often so profound as to render both sides of the body equally irresponsive. In this event a valuable aid to the diagnosis is the Marie-Foix¹ reaction. This test is made by forcibly pressing the foot into extreme plantar flexion and at the same time compressing the toes. On the paralyzed side a sharp contraction will be observed in the tensor vaginæ femoris and quadriceps extensor muscles. The same procedure on the unparalyzed side causes little or no response.

When it is clear that a hemiplegia is present the next step is to determine, if possible, whether it is caused by hemorrhage, embolism, or thrombosis. The blood pressure furnishes important and significant information in this connection. If the pressure is high the inference may be made that the cause of the apoplexy is hemorrhage or embolism. Auscultation of the heart sounds is the next step and if no cardiac murmurs are heard the case may be provisionally regarded as one of hemorrhage. The state of consciousness of the patient should be noted, and, if unconscious, the depth of the coma estimated as shown by the condition of the pupils and their reaction to light. As collateral evidence the character of the patient's activity when stricken should be considered and the question of prodromas inquired into when such information is available. Contrary to generally accepted ideas, prodromal symptoms are more frequent in thrombosis and embolism than in hemorrhage.

Before arriving at a tentative diagnosis of cerebral hemorrhage, certain other possibilities must be considered. Chief among these is syphilitic endarteritis of the brain. Cerebral neoplasm, abscess, and meningitis should also be thought of as possible causes of the hemiplegia. These topics are dealt with *in extenso* in other chapters and hence only such phases of them as have bearing on the immediate diagnosis of cerebral hemorrhage in its early stages will be mentioned here. In case of an adult under forty years of age, found comatose and with a demonstrable hemiplegia, the question of syphilitic endarteritis should be raised at once. It may be adequately settled only by subsequent examination of the blood for the Wassermann reaction. In many

¹ See article by Jelliffe: Little Signs of Hemiplegia, Postgraduate, October, 1912.

cases of this type in which this reaction cannot be obtained from the blood, it is found present in the cerebrospinal fluid. Other evidence of a luetic condition should be sought for. In rare cases coma and hemiplegia have been reported as occurring in the secondary stage of syphilis. Wherever possible it is always advantageous to make an ophthalmoscopic examination for the patient's eye-grounds. Any of the electric ophthalmoscopes are serviceable for this purpose. If the pupillary contraction is excessive a very convenient form of mydriatic is the cocaine hydrochlorate tablet of Burroughs & Wellcome, each containing $\frac{1}{80}$ gr. of the drug. Two of these in either eye will give a satisfactory dilatation in five or ten minutes. The presence of a diffuse retinitis or a retrobulbar neuritis should still further strengthen the suspicion of a syphilitic factor in the disease. Ophthalmoscopic examination is valuable in all cases. In cerebral hemorrhage the vessels of the optic disk are engorged and the disk itself after a relatively short time, in many cases, shows a distinct tendency toward cupping. This is seldom true in thrombosis or embolism. Cerebral neoplasms in 85 per cent. of all cases show a distinct choked disk in one or both eyes. Here, again, the blood pressure will aid in the diagnosis. In cerebral neoplasm, syphilitic endarteritis, and abscess the blood pressure is relatively low. It is uniformly high in cerebral hemorrhage, is generally high in cerebral embolism and low in thrombosis. Coma and hemiplegia in a patient with high blood pressure (190 mm. or above) engorgement of the vessels of the optic disk, and a tendency toward a papillary edema, constitute presumptive evidence of a cerebral hemorrhage.

After a provisional diagnosis of hemorrhage has been made the first consideration influencing the treatment is the place in which the patient has been stricken and what immediate disposition must be made of him. If the apoplectic attack has occurred in some place other than the patient's home the effort should be made to accommodate him temporarily without removal. If he has fallen unconscious upon the floor he should be allowed to remain there and moved as little as possible. The head and shoulders should be elevated by one or two pillows, and all tight clothing removed, especially in the case of women. Care must be taken not to move the patient about unnecessarily and for this reason it is best to cut the clothing. If it is necessary to transport the patient to hospital or home the utmost care must be exercised. The patient should be cautiously placed on a stretcher with the head and shoulders slightly elevated and transferred to the vehicle with as little jarring or swinging as possible. The same precautions must be taken in placing the patient in bed. The head of the bed should be elevated to an angle of 10 degrees or 15 degrees and the blood pressure again taken. If it has increased decidedly and if the coma has become more profound the advisability of phlebotomy should be considered. In all cases in which the blood pressure steadily rises and tends to exceed 250 mm. of Hg. beneficial results are had from opening a vein and drawing off 10 to 12 ounces of blood. Leszynsky reports a case of a man suffering from cerebral hemorrhage in profound coma with a

systolic blood pressure of 300 mm. After 14 ounces of blood were removed the pressure fell rapidly to 190 mm. and the patient regained consciousness. Phlebotomy is a procedure to be resorted to only in cases of excessively high pressure and deep coma.

It frequently happens that the patient is stricken in some part of his home remote from his sleeping quarters. In such cases no attempt should be made to remove him to his own bedroom, but temporary accommodations must be provided for him in the place where he has fallen. After the clothes have been removed, as already suggested, and the patient placed in bed a hot mustard foot bath or hot packs over the lower extremities will help in reducing the blood pressure. In some cases where the hemorrhage is evidently extensive and long continued a tourniquet about both legs near the trunk is of service. This seems to be of particular value in hemorrhages of the ingravescent type. An ice cap placed upon the head is usually recommended and may have some efficacy.

The bladder should be especially watched and the patient catheterized at once if there is any considerable amount of urine present. This particular should be a matter of special care for the first few days following the attack, or so long as the coma lasts

Hypodermic Medication.—Medication at this time has one chief object, the reduction of the blood pressure. The drugs prescribed should be given by mouth preferably, but in many cases this is dangerous, since the patient cannot swallow or has some degree of dysphagia. Hypodermic injection must then be resorted to

DRUG ACTION ON BLOOD PRESSURE.—Several years ago the writer had the opportunity to conduct a series of clinical experiments, with the object of testing the value of certain cardiovascular agents, administered hypodermically, in their application to cerebral hemorrhages. Hemiplegic patients were selected for these tests and observations of the blood pressure were made at intervals of every five minutes after the hypodermic injection of the drug. The experiments were conducted in groups of five to ten patients each and an average pressure curve was computed from the readings obtained. In all 150 patients were examined under these conditions. The majority of these patients presented a marked elevation of blood pressure as one of the symptoms of their diseased conditions.

Distilled Water.—The effects of hypodermic injection of distilled water showed that in every instance there was a decided rise of blood pressure.

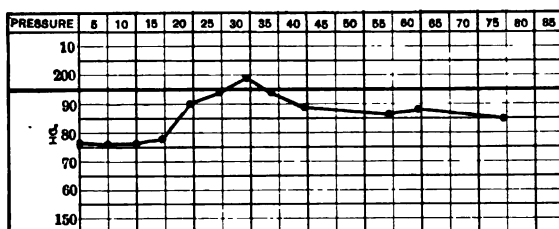
AVERAGE WATER CURVE (AQUA DESTILLATA, M_x), AVERAGE INITIAL PRESSURE 180.2 MM. HG.

Interval.	Pressure.	Interval.	Pressure.
End of 5 min.	180.2 mm. Hg.	End of 35 min.	197.2 mm. Hg.
End of 10 min.	180.2 mm. Hg.	End of 40 min.	193.4 mm. Hg.
End of 15 min.	181.2 mm. Hg.	End of 55 min.	190.2 mm. Hg.
End of 20 min.	195.0 mm. Hg.	End of 60 min.	192.6 mm. Hg.
End of 25 min.	199.0 mm. Hg.	End of 75 min.	190.6 mm. Hg.
End of 30 min.	205.0 mm. Hg.		

This curve indicates that the injection of plain water causes an elevation in the blood pressure and suggests the necessity, when employing any depressant, of selecting such preparations and strengths as will not only efficiently reduce the pressure, but even more effectually overcome the elevating effects of the water.

Aconitine.—The first depressant employed in the experiments was aconitine, the active principle of aconite. Hypodermic tablets marked "Aconitine (Pure Crystal) grain $\frac{1}{20}$," and prepared by two standard tablet manufacturing houses, were used. The injection solution (aqueous) was so made up that every ten minims represented $\frac{1}{20}$ grain of aconitine.

FIG. 36



Action of aconitine (crystal).

In every case the blood pressure rose after a very short interval, the maximum elevation being 35 mm. Hg., the minimum being 5 mm. Hg. The interval elapsing between the time when the rise first became apparent until it reached its summit varied from ten to twenty minutes, being on the average about fifteen minutes. After the primary elevation there followed, as a rule, a subsequent fall in pressure.

Computation of the figures obtained from the ten individual curves charted for aconitine, grain $\frac{1}{20}$, furnishes the basis for the following average pressure curve:

AVERAGE PRESSURE CURVE; ACONITINE, GRAIN $\frac{1}{20}$. (HYPODERMIC TABLETS MARKED "PURE CRYSTAL.")

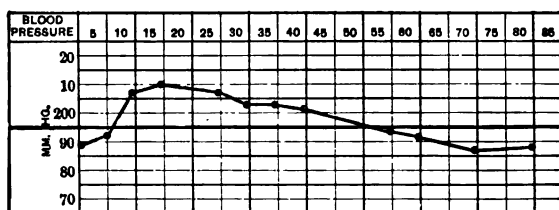
Interval.	Pressure.	Interval.	Pressure.
End of 5 min.	196.25 mm. Hg.	End of 35 min.	208.75 mm. Hg.
End of 10 min.	212.50 mm. Hg.	End of 40 min.	205.0 mm. Hg.
End of 15 min.	215.25 mm. Hg.	End of 55 min.	199.75 mm. Hg.
End of 25 min.	212.75 mm. Hg.	End of 60 min.	196.25 mm. Hg.
End of 30 min.	208.75 mm. Hg.	End of 70 min.	193.0 mm. Hg.
		End of 80 min.	193.5 mm. Hg.

Aconitine (Potent Crystal) in doses of grain $\frac{1}{20}$ gave results which, if not altogether satisfactory, at least demonstrated that the drug in this preparation and dosage is a somewhat active hypodermic agent for reducing blood pressure. Its effects were not always uniform and therefore decidedly open to objection.

In two instances the curve described a distinct primary rise, followed

by a subsequent fall, which in turn gave place to a secondary rise, carrying the curve within a few points of the initial pressure. This phenomenon was so at variance with the general character of the other observations that an attempt was made to explain its appearance upon some basis other than the drug influence, but nothing which would tend to explain the exceptional character of the curve in the two instances cited could be brought to light. It was therefore decided to allow them to stand as representative of the experiment. The effect of these two primary elevations upon the average curve is, of course, only fractional, but must be assigned its due value.

FIG. 37



Action of aconitine (potent crystal, Merck).

Computation of the figures obtained from the ten individual curves charted for aconitine, grain $\frac{1}{150}$, furnishes the basis for the following:

AVERAGE CURVE (ACONITINE—POTENT CRYSTAL, MERCK), GRAIN $\frac{1}{150}$, INITIAL PRESSURE 171.5 MM. HG.

Interval.	Pressure.	Interval.	Pressure.
End of 10 min.	170.4 mm. Hg.	End of 50 min.	156.3 mm. Hg.
End of 15 min.	167.0 mm. Hg.	End of 55 min.	165.5 mm. Hg.
End of 20 min.	167.7 mm. Hg.	End of 60 min.	165.9 mm. Hg.
End of 25 min.	167.1 mm. Hg.	End of 65 min.	169.6 mm. Hg.
End of 30 min.	165.2 mm. Hg.	End of 70 min.	165.2 mm. Hg.
End of 35 min.	155.9 mm. Hg.	End of 75 min.	168.2 mm. Hg.
End of 45 min.	154.7 mm. Hg.	End of 80 min.	172.7 mm. Hg.
		End of 85 min.	178.9 mm. Hg.

Considered *per se* this accredits aconitine, grain $\frac{1}{150}$, with a slight primary depressant effect. At the end of ten minutes the pressure is reduced a little over 1 mm. Hg. In 15 minutes the total reduction is 4.5 mm. Hg., at about which level the curve remains for the ensuing fifteen minutes. Then occurs a still greater decline, carrying the curve downward for a gross drop of 17 mm. Hg. At the end of fifty minutes the curve begins to ascend again until in eighty-five minutes it reaches a point 7 mm. Hg. higher than the average initial pressure.

Gelseminine Hydrochlorate.—Gelseminine hydrochlorate in doses of $\frac{1}{25}$ grain acts as a mild depressant. Its primary depressant effect is followed by a slight rise, with a subsequent secondary decline which tends to be more persistent. In one case, instead of a primary

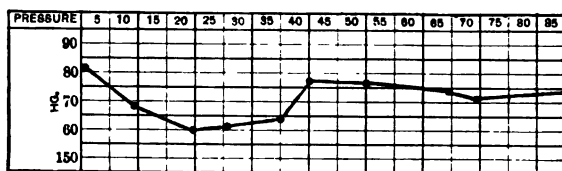
decline a decided primary rise appeared. This primary rise could not be explained upon any tenable grounds and therefore figured in the determination of the average curve. Computation of the figures obtained from the five individual curves charted for gelseminine hydrochlorate, grain $\frac{1}{15}$, furnishes the basis for the following:

AVERAGE CURVE, GELSEMININE HYDROCHLORATE, GRAIN $\frac{1}{15}$, AVERAGE INITIAL PRESSURE 186.7 MM. HG.

Interval.	Pressure.	Interval.	Pressure.
End of 5 min.	186.7 mm. Hg.	End of 50 min.	196.0 mm. Hg.
End of 10 min.	185.28 mm. Hg.	End of 55 min.	196.0 mm. Hg.
End of 15 min.	185.28 mm. Hg.	End of 60 min.	189.0 mm. Hg.
End of 20 min.	182.18 mm. Hg.	End of 65 min.	167.2 mm. Hg.
End of 25 min.	190.58 mm. Hg.	End of 70 min.	166.1 mm. Hg.
End of 30 min.	196.0 mm. Hg.	End of 75 min.	166.2 mm. Hg.
End of 35 min.	196.0 mm. Hg.	End of 80 min.	166.3 mm. Hg.
End of 40 min.	196.0 mm. Hg.		

Gelseminine hydrochlorate was also used in doses of $\frac{1}{15}$ grain; this exceeds the dosage generally recommended by the authorities, but no untoward results from its use were observed. At the same time it proved the most reliable and consistent agent employed. Its first effect is to produce an early and marked decline in the blood pressure.

FIG. 38



Action of gelseminine hydrochlorate.

This depression is maintained for some time, whereupon, as in all other cases, a subsequent return to the initial pressure occurs.

AVERAGE CURVE, GELSEMININE HYDROCHLORATE, GRAIN $\frac{1}{15}$, AVERAGE INITIAL PRESSURE 185 MM. HG.

Interval.	Pressure.	Interval.	Pressure.
End of 10 min.	175.0 mm. Hg.	End of 65 min.	179.4 mm. Hg.
End of 20 min.	165.0 mm. Hg.	End of 70 min.	173.4 mm. Hg.
End of 25 min.	165.0 mm. Hg.	End of 100 min.	179.6 mm. Hg.
End of 35 min.	167.6 mm. Hg.	End of 110 min.	175.6 mm. Hg.
End of 40 min.	183.6 mm. Hg.		
End of 50 min.	182.6 mm. Hg.		

Aconitine in the commercial form of hypodermic tablets is not to be relied upon, its action oftentimes being the direct opposite to the one desired. In solution aconitine (potent crystal, Merck) in doses as high as $\frac{1}{150}$ grain is not a desirable agent when a rapid, reliable

and decided depressing effect is needed. Gelseminine hydrochlorate in doses of $\frac{1}{16}$ grain produces a rapid and marked decline in blood pressure and, therefore, when compared with aconitine seems to be a more useful and reliable agent. Its depressing effect, however, is not long sustained. Whether it will prove more stable in tablet form than appears to be the case with aconitine is still a question.

The significance and dangers of the early return to the initial pressure, uniformly observed after the use of any of the agents employed, are evident enough without explanation. That such an elevation would become a crux in dealing with cases of cerebral hemorrhage cannot be denied, the only question being how to prevent it. The suggestion offered is the continuing injection of the drug solution at the time when, according to the average curve, the secondary elevation first makes its appearance.

The dangers and disadvantages of the hypodermic mode of administration for cardiovascular depressant effects are well illustrated by what has been called the water-curve type. It is evident that in aqueous hypodermic solutions of any depressant agent the drug must be of such strength as to overcome the primary stimulating effect of the water, while the water itself must be present in such small quantities as to cause the least possible amount of stimulation. In other words, aqueous hypodermic solutions for depressing effects must be as highly concentrated as possible. The use of cardiovascular depressants dissolved in a hypodermic cylinder full of water (m20 to 30) is to be avoided.

USE OF NITROGLYCERIN OR NITRITES.—The use of nitroglycerin or nitrite preparations cannot be recommended, since these agents produce a marked initial elevation of blood pressure and are all alike evanescent in their depressant effects.

Medication by Mouth.—As already stated, it is advisable to administer drugs by the mouth, provided the patient is able to swallow. Aconite is the most reliable of the cardiovascular depressants. It is best given as the tincture, although some authorities object to its use in this form because of the contained alcohol, which they regard as a possible stimulant, and hence contraindicated under the conditions. To meet this difficulty the fluidextract may be substituted. The following doses of the different pharmacopœial tinctures may be given.

Tincture of aconite	U. S. P. (1900) m5
Tincture of aconite	U. S. P. (1890) m1
Tincture of aconite	B. P. m5

The interval between doses as well as the size of the dose will depend upon the influence the drug exerts upon the blood pressure and the susceptibility of the patient to its toxic properties. If an initial dose of 5 minims has produced no effect upon the arterial pressure, this dose should be repeated at the end of an hour. With a pressure of 200 mm. or over, it is safe to start with a dose of 10 minims of the tincture, but in any event the blood pressure must be taken at least

every half-hour. Should the first dose produce no marked change in the pressure the next one must be increased. Administration in this way may be continued for several hours or until the desired depressant effect is obtained. The general condition of the patient must be carefully watched lest the drug cause a dangerous toxemia. Indications for the discontinuance of the aconite are dilatation of the pupil, vomiting, irregularity of the pulse, cold moist skin, especially over feet and hands, an erythematous rash, and dyspnea. If the fluidextract is the form selected, its administration should be even more carefully supervised. It may be given in doses of $\frac{1}{2}$ to 2 minims every hour until the blood pressure is sufficiently decreased.

The question naturally arises as to what constitutes a sufficient decrease of the blood pressure. The answer to this will depend entirely upon how high the arterial pressure was before the administration of any drug. In the average case a fall of 20 to 30 mm. may be deemed sufficient, but in all cases with an initial pressure well above 200 mm. the attempt must be made to bring this down below the 200 mm. mark.

Once having reduced the blood pressure to a sufficient degree, it will be necessary to hold it at this or a lower level. This may be accomplished by continued smaller doses of aconite at the same intervals, or the original dosage at longer intervals.

Veratrum viridi may be substituted for aconite. Its use is more especially indicated in cases complicated by uremia. The fluidextracts should be given in doses of 2 to 5 minims every hour or the tincture in doses of 5 to 10 minims at the same interval. As in the case of aconite the size of the dose as well as the length of the interval between doses will depend upon the manner in which the blood pressure responds to the drug.

Gelseminine is another useful substitute for aconite. It may be administered as the fluidextract in doses of 5 to 10 minims or as the tincture in doses of $\frac{1}{2}$ to 1 dram.

Apocynum in the form of the fluidextract in doses of 5 to 20 minims is also a valuable depressant.

In the event of using any of the cardiovascular depressants, either by mouth or hypodermically, the blood pressure should be taken every two hours and should serve as the chief guide to the frequency and size of the dose given. Nitroglycerin and the nitrite by mouth should be avoided in all cases. They produce a primary elevation of blood pressure while their depressant effects are very fleeting.

PURGATIVES.—Early and active purgation of the patient is especially recommended. If deglutition is not too difficult and imperfect a compound cathartic pill should be given as soon as possible after the patient has been placed in bed. Should the coma be profound, one drop of croton oil (*oleum tigllii*) may be applied to the back of the tongue.

SEDATIVES.—In cases presenting convulsions, delirium, or restlessness, bromides, chloral, or hyoscine hydrobromate will prove useful. If the patient be alcoholic small doses of whisky may be required.

Patients often suffer from severe retching, vomiting, or hiccough. Under these conditions the stomach should be washed out at once. In this lavage it is most convenient to pass the tube through the nose. Should this means fail to give relief a hypodermic of morphine sulphate, grain $\frac{1}{8}$, and atropine sulphate, grain $\frac{1}{120}$, may be given and repeated in an hour if necessary. The general object of sedatives under all circumstances is to prevent unnecessary physical exertion on the part of the patient.

Feeding and Diet.—Ordinarily the patient may be allowed to go without food for forty-eight hours. At the end of this time fluid nourishment may be given by mouth if the patient is able to swallow, but if a marked degree of dysphagia is present rectal feeding should be resorted to. It is essential in all cases to make certain that the patient can swallow and always to begin mouth feeding through a long-tubed dropper, and later by a spoon introduced well into the mouth. In this way the dangers of aspiration-pneumonia and coughing spasms may be avoided.

The diet for the first week should be purin-free, consisting of milk and gruels. Tea, coffee, cocoa, and all malt liquors must be interdicted. Sugar and eggs may be added to the milk and gruels.

Six to eight ounces should be given at each feeding, at intervals of three to three and a half hours. Particular care should be taken that the feeding is slow and under no circumstances given out of an ordinary glass or cup during the first day or two. Always feed by spoon or dropper until the patient is able to swallow with some degree of ease.

While the patient remains in bed his diet should be low in purins. All meats (fish, fowl, game) except sole and codfish must be excluded. This also applies to peas, beans, asparagus, and onions. For the sake of variety, selection may be made from potatoes, turnips, carrots, rhubarb, spinach, milk, eggs, cheese, butter, sugar, white bread, rice, tapioca, cauliflower, lettuce, and macaroni. Some of the various artificial animal or vegetable protein foods may also be used.

In cases of glycosuria all carbohydrates must be withheld.

Artificially soured milk is often beneficial if an indicanuria is present.

Cases giving evidence of a chronic parenchymatous nephritis do well on Widal's salt-free diet.

If the patient is unable to swallow it will be necessary to feed by rectum after forty-eight hours. It should be remembered that 500 calories per day is about the limit of absorption from this mode of feeding. Milk is commonly the basis of enemas, and the following are useful combinations:

- 1 { Peptones, 60 grams.
Milk, 200 c.c.
- 2 { Eggs, 3 grams.
Salt, 3 grams.
Milk, 250 c.c.
- 3 { Grape sugar, 60 grams.
Milk, 250 c.c.
Starch (unboiled), 60 grams.
Milk, 270 c.c.

In administering a nutrient enema the following directions should be observed.

1. Quantity of each enema, 9 ounces (250 c.c.).
2. The tube used should be small and of soft rubber, which must be introduced at least twelve inches in the rectum.
3. The distance through which the fluid is allowed to fall should be three feet.
4. Three to four enemas should be given daily.
5. One cleansing enema should be given each day.
6. If the rectum is intolerant and refuses to retain the fluid a small amount tincture of opium may be added to each enema and the fluid should be allowed to flow in slowly.

Regime of the Sick Room.—The sick room must be kept as free from all disturbing elements as possible. After the patient has regained consciousness care must be taken to avoid emotional excitement. Friends and family must be made to understand that their solicitude should not be expressed in the sick room. The efforts of attention and attempts to participate in conversation with others than those in immediate attendance are always a serious tax on the patient. During the first week it is not advisable for more than one member of the family to be in the sick room at a time. The nurse in charge should be instructed to enforce this regulation. The temperature of the room should be kept as nearly fixed as possible and free ventilation provided. In winter the patient must not be exposed to draughts or sudden changes of temperature so frequently produced by opening the windows wide in order to air the room. This will not be necessary if the temperature and ventilation are made matters of routine care. In summer, when the heat is excessive a small electric fan will contribute greatly to the comfort of the patient and lessen the dangers of heat prostration so commonly seen in these cases.

The Bed.—If a choice is possible, the bed should be a single one. The mattress and bedding must be arranged to prevent sagging in the middle, and so form a deep depression into which the patient sinks. This may be in part obviated by a board placed between the springs and the mattress at about the middle of the bed. In protracted cases, and particularly for aged people, a special type of invalid bed is convenient and reduces the difficulties of handling the patient. A very satisfactory device of this kind is the Wallace Invalid Bed. This provides several desirable changes in the bed surface and is easy to manage.

Care of the Gastro-intestinal Tract.—The bowels should be kept freely open by the use of appropriate cathartics. One evacuation is necessary every day. Two or even three movements are needed if this has been the patient's habit before he was stricken. In case of diarrhea or too frequent evacuation bismuth may be employed. A useful combination for this disorder is the following.

R—Tannogen	gr. v
Heroin	gr. $\frac{1}{16}$

This preparation may be repeated every hour for four doses. If medication by mouth is impossible, the enema used to cleanse the bowel prior to feeding will usually suffice for emunctory purposes.

The mouth and teeth should be kept clean. Some mildly acid mouth-wash is especially useful. In some cases excessive salivary secretion becomes a menace to the patient. This is especially true in cases of deep coma. Under these circumstances it will be necessary to swab the mouth out continually. Small doses of atropine or tincture of belladonna will inhibit the salivary secretion.

In order to prevent hypostatic pneumonia the patient may be rolled over on the sound side three or four times a day. This should not be attempted, however, before the second or third day.

Care of the Skin.—Especial attention must be given to the skin. Some cases develop bed-sores with remarkable rapidity. The first requisite is to keep the skin dry and well powdered, particularly about the buttocks. Pads of absorbent cotton may be applied to the external genital organs and changed as soon as wet or soiled. The treatment of a bed-sore when such has once developed will often be a matter of great difficulty. Antiseptic dusting powders seem to be more beneficial than ointments. Efforts should also be made to relieve the area involved of the superincumbent weight of the body. This may be accomplished by the use of air cushions or rings well padded. In extreme cases it may be necessary to place the patient on a water-bed. The patient should remain in bed for at least three weeks and longer if the resulting paralysis is profound and the blood pressure remains high.

Care of the Paralyzed Limbs.—It is advisable to apply a light wire anterior splint to the paralyzed arm while it is still flaccid. In this way the tendency to contraction when the spasticity begins will be, to some extent, prevented. If these splints are used they should be removed at least once in twenty-four hours, the limb freshly powdered and wrapped in cotton before reapplying the splint. The further care of the paralyzed limbs is given more in detail in the following pages.

In recent times some operative procedures have been suggested for the relief of cerebral hemorrhage. Among these are trephining, brain puncture, decompression for drainage, compression or ligation of the internal carotid arteries. These operations have not met with general approval. On the other hand, nephritic patients suffering from cerebral hemorrhage with stupor and convulsions do well as a result of lumbar puncture and the withdrawal of from 25 to 50 c.c. of cerebrospinal fluid. Nephritic patients also improve remarkably as a result of phlebectomy. The presence of nephritis adds a complication to the cerebral lesion, which demands special treatment of its own.

Treatment of the Period of Paralysis.—The treatment of this period should have three main objects: (1) The reconstruction of the impaired motor functions as much as this may be, or the substitution of other muscular acts for those impaired or lost; (2) the combating of certain

sequelæ and complications; and (3) the prevention of a recurrence of the apoplectic insult.

The real efforts toward exercising the paralyzed limbs or preventing deformities cannot proceed far until the patient has been gotten out of bed. The matter of getting the patient up deserves considerable attention. With elderly and feeble people the process of getting up should be more gradual than in the case of those who are robust and naturally vigorous. During the second week, if there are no indications to the contrary, the patient should be put in a nearly sitting posture for two or three hours at a time. Care should be taken that the legs are not allowed to remain extended, but are brought into flexion and supported in this position by a pillow or cushion. Once accustomed to this position the patient should next be allowed to sit on the edge of the bed in such a manner that the feet rest on the floor. In this position he may take his meals. The ordinary method of "sitting a patient up in bed" must be avoided. Nothing can be more uncomfortable than this posture, for the full extension of the legs on the thigh not only causes an unnatural position of body, but by producing an unusual strain on the muscles will prove fatiguing. If the patient is to sit up at all, let him do so in a natural position. Generally by the end of the third or fourth week a wheel chair should be used and the patient may be allowed to sit up the greater part of the day..

Arthritis of Shoulder-joint.—Prominent among the complications of hemiplegia is arthritis of the shoulder-joint, resulting from the abnormal position and articular pressure of the paralyzed arm. This same affection may occur in the wrist, elbow, ankle, and hip. To avoid this condition passive movements should be commenced at the end of the second week; they should not be continued longer than two or three minutes at each sitting. The time given to these movements as well as their extent should be increased from week to week. Massage, especially of the muscle groups about the joint in question, must be given in conjunction with passive movements. Even when a mild degree of arthritis is present much can be done to correct the condition by the means just mentioned. If the arthritis remains untreated, ankylosis often results and the trouble then requires more active measures. Under these circumstances Lewandowsky has found the injection of fibrolysin to be of much help in combination with massage and passive movements.

Contractures.—Contractures are another source of difficulty complicating the period of paralysis. They usually affect the large flexor groups of the extremities and also the adductors. It is, no doubt, the disparity in strength between the flexors and extensors that determines the contractural positions as a result of the hypertonic condition of the muscles. Much may be accomplished in the prevention of such occurrences by early fixation with light splints which are so arranged as to oppose the muscle groups causing a contracture. The greatest difficulty will be experienced in correcting and preventing the flexures of the fingers. A light anterior splint should be employed which will

extend beyond the ends of the distal phalanges and well up to the middle of the arm. If the hypertonus becomes marked it will be necessary to supplement this fixation by a shorter dorsal splint. The leg should also receive attention, for it is the neglect of these points in the early stages of the paralysis that often leads to unnecessarily severe defects in gait and motion generally. While the patient is still confined to the bed, the weight of the bed-clothing should be kept off of the foot by means of any of the arched frames; the foot should be so supported or strapped as to prevent the usual tendency toward plantar flexion and external rotation. In the event that the increasing spasticity causes the leg to be flexed at the knee it is expedient to apply light traction to produce extension or even to immobilize the knee with the leg extended by a light plaster of Paris cast. There can be little doubt that the more serious disturbances in the gait and contractures of the arm, such as one sees in the large public hospitals, have been favored by the lack of attention of these details in the early treatment.

Tenotomy.—Tenotomy has been advised to relieve the deformities produced by the contractures. This has its distinct indications in certain cases, especially when the spasticity cannot be overcome by any of the other means. It is particularly useful in the treatment of Soleus-gastrocnemius contractures, but it is questionable whether the advantage to be gained in treating contractures of the fingers is sufficient to warrant the operation. All of these procedures must be carried on with the utmost care, for it is an easy matter to occasion a decubitus by splints, casts and the like in tissues which are so nearly defenceless as the paralyzed extremities.

Section of Dorsal Spinal Roots.—Section of the dorsal roots of the spinal nerves has been advocated as a means of combating contracture or the tendency toward it. It is, however, a nice question to decide whether the contracture is due to hypertonus of the muscle or actual shortening in the muscle substance. If the former can be proved to be the case dorsal root section may be considered, but not, however, until other less radical means have failed to meet the conditions.

Painful Cramps.—Cramps of a painful and distressing character often appear as a complication in the disease. This is especially true in cases of pseudobulbar palsy in which the apoplectic attacks follow each other rapidly, involving one side of the body and then the other. Such cases do best without massage and electric treatment. The relief of the pain is usually well accomplished by codeine and acetanilide in combination, but it may be necessary to resort to morphine. For the actual improvement of this condition the method which has proved most uniformly successful is extension by means of weights, beginning with light traction for a short time and gradually increasing to longer periods and heavier weights.

Electricity.—Electricity, in several respects, may prove a valuable accessory to the treatment. The galvanic current is indicated in those relatively rare cases of hemiplegic pain. In applying the current for this purpose the anode should be used over the painful areas. In some

cases a marked atrophy of the arm, and less frequently of the leg, occurs; here the galvanic or the galvano-faradic currents are useful. The best current modification in these cases is the sinusoidal movement. Patients can in this way tolerate a greater number of milliampères than they will without this modification. The galvanic sinusoidal current will prove helpful as an aid to certain exercises to be described later. Some authorities recommend the high tension current for the reduction of blood pressure. The value of electricity in any form must not be lost sight of. In nearly all instances it has an excellent psychic effect and furnishes a motive which is helpful in the management of the case. In using electricity its limitations should be recognized. It should only be recommended as an auxiliary to the treatment. It can have no direct or curative effect upon the paralysis of a cerebral hemiplegia. The most generally useful currents are the galvano-faradic, sinusoidal current, and the faradic with a relatively high rate of interruption.

Active Movements.—At the end of three or four weeks the patient should be advised to begin sustained active movements. If there are no indications to the contrary he may be kept up in a chair the greater part of the day and frequent attempts should be made at walking. Every movement that remains or has returned to the arm and leg should be repeated many times to the end that each action will gain steadily in execution. Each returning movement should be added to the repertoire of acts to be practised. When the patient is at length able to walk with some degree of certainty mild gymnastic exercises are of value. If the motor functions involved in speech have been disturbed with a resulting aphasia, the attempt should be made to reëducate the patient in spoken language and while doing so insist on the use of the left hand in writing. If the patient is naturally left-handed he should then be instructed to use his right hand in writing. The amount of restored motor function which may be expected of these means will be proportional to the size and location of the hemorrhages. In some cases of slight bleeding encroaching but little upon the posterior limb of the internal capsule the improvement is remarkable, amounting almost to complete recovery. In the severe cases only slight gain will be made, irrespective of the methods of treatment employed.

Prevention of Recurrence of Attack.—The third object of the treatment during the period of paralysis is the prevention of a recurrence of the apoplectic attack. With the intelligent coöperation of the patient this will meet with a fair measure of success. How much may be expected or promised in this regard must depend entirely upon the nature of the case and the degree of involvement. The blood pressure during this period is, almost without exception, high, and is, for this reason, a constant menace. Therapeutic means of combating this condition have not proved satisfactory. The use of the usual cardiovascular depressants has the double disadvantage of disturbing the digestion and producing only transitory depression of the blood pressure. Some writers are strongly inclined to consider the high blood pressure

observed in the period of insult as well as in the period of paralysis the result of direct pressure effects upon the brain tissue. This view seems untenable in the light of the fact that the blood pressure is nearly always high in the period of preparation, before any direct pressure from hemorrhage has occurred. It is far more likely that the high arterial tension is fundamentally due to general systemic changes in the blood-vessels. The treatment for preventing the repetition of the cerebral hemorrhage differs little from that advised for the prevention of the initial apoplexy.

The gastro-intestinal tract should receive particular attention. The bowels should be kept active and the tendency to constipation met by cathartics. Drastic cathartics should be avoided. The drugs and dosage employed must be so selected for the individual case as not to produce too active purgation or hydragogue catharsis. Enemas and colonic irrigation will prove the safest and most efficient means of dealing with obstinate constipation. The use of colonic irrigation should be controlled by observation as to the blood pressure. In some cases the pressure will rise perceptibly during the irrigation and remain elevated for a considerable time. Under such circumstances colonic irrigation is contraindicated. Evidence of gastro-intestinal toxemias should be watched for. Gastric indigestion with the production of flatus must be corrected by the closer regulation of the diet and the use of appropriate carminatives.

The subject of the diet is here as everywhere a question of exceptions rather than the rule. The necessities of each individual case must be studied and a diet selected which shall have the merits of causing little or no gastric disturbances and no enteric toxemia so far as may be determined by the analysis of the stool and urine. This regulation may require, in some instances, a partially predigested diet. The urinalysis will indicate to what degree the proteins or carbohydrates must be restricted. Not a few cases present a marked glycosuria for some time after the insult and should be treated accordingly. The presence of large amounts of indican in the urine is often not as significant of indolemia as the appearance of this substance in small amounts. It is not necessary to wait for indican to appear in large quantities before taking steps to prevent indolemia. Catharsis, irrigation if applicable to the given case, restriction of protein foods and a gradual increase of exercise where possible will prove valuable to this end. The addition of some of the artificially soured milks to the diet is also advisable under these conditions.

General Hygiene.—In the readjustment of the patient's habits following the apoplectic attack, many questions concerning the general matters of hygiene will arise. The amount of exercise which should be permitted will depend in great measure upon the degree of the paralysis. It cannot be considered a good practice to forbid all exercise, but such activity as is recommended should be so directed that the patient is allowed by slow stages to carry on sustained muscular acts. Wherever circumstances will permit the exercise should be taken out

of doors. This may not be possible for several months after the seizure. The patient should not be allowed to climb stairs more than possible and should be instructed to rest frequently in doing so. As time goes on the outdoor exercise may be increased to fairly long walks. Emotional disturbances, anxiety, and other annoyance should be avoided. It is well to impress upon the patient that to make the most of the functions which remain to him he must limit his activities in every way possible. The stress of business, the responsibilities of official positions, the active participation in family affairs cannot fail to prove harmful and perhaps disastrous. If the circumstances permit the case should be fairly stated to the patient and the possibilities both of his condition and of the treatment should be outlined. The emotional state, amounting oftentimes to moral panic, which follows the patient's realization of the nature of his disease, must be combated by all in attendance, nor must despondency over an apparently hopeless position be allowed to become the controlling influence. All of these details are matters intimately involved in the personal equation, and yet the neglect of any of them will seriously interfere with the success which may be hoped for from the treatment.

Baths and cures of various kinds have been recommended as beneficial during this period; but like all other therapeutic agents for this purpose these means do not yield uniformly good results. Some patients, especially of the nephritic class, do well under this kind of treatment, but it must be borne in mind that any therapeutic measure which causes a temporary or permanent elevation of blood pressure is distinctly contraindicated.

Exercises for the Reëducation of the Paralyzed Limbs.—This feature in the treatment of the paralysis is too frequently slighted. In consequence many severe deformities are permitted to develop which almost completely incapacitate the patient. On the other hand, the proper application of the principles of exercise requires more nearly ideal conditions than usually obtain. The physician must not only understand the anatomical defects with which he is dealing but he must know exactly to what end he is directing the exercises to be employed. He must further be assured that those in attendance upon the patient are capable of carrying out the directions given and are faithful in doing so. Indefinitely prescribed exercises are of little avail and are soon discontinued. If any good is to be obtained from physical exercise it will only be the result of persistent effort and constant encouragement.

The first requisite in instituting this line of treatment is to enlist the interest of the patient and so secure his coöperation. The idea that effort may successfully be made to restore, in some degree, the lost functions will serve to arouse his enthusiasm and inspire his confidence. Too frequently these invalids are overwhelmed by the sense of their own catastrophe and are allowed to sink into a state of apathy and dejection. There should be a distinct understanding between physician and patient as to just what benefits may reasonably be

expected from the treatment, as well as to the slow nature of the improvement and the necessity of systematic, unremitting endeavor.

The types of movements which may be employed for physical exercise are six in number, *i. e.*

1. Passive movements.
2. Movements against which the patient makes resistance.
3. Voluntary movements in which the patient is assisted by an attendant.
4. Voluntary movements assisted by electricity.
5. Voluntary movements which the patient supplements by some mechanical appliance.
6. Unassisted voluntary movements.

Passive movements should be commenced as soon as the patient regains consciousness. They should then be very limited in extent and of short duration. The muscles at this time are still flaccid so that there will be but little hypertonic resistance to work against. Special attention should now be given to the hand and fingers, foot and toes. The precaution of splinting having already been taken, the fingers and hand should be forcibly extended each time the splint is removed. This should be repeated five or ten times on each of these occasions. The toes and foot should receive similar attention. At the end of the first week the forearm and leg should be included in these passive movements, provided the patient's condition in other particulars does not contraindicate these manipulations. By the end of the second week the patient may be instructed to resist the passive movements, and even though no actual resistance is possible the attention should be directed and held to this purpose.

The supervision of the active, voluntary movements should be guided by the character and extent of the muscular disability generally observed in hemiplegics. Not all of the muscle groups in the leg and arm are involved to the same degree. It is a rule, with but few exceptions, that the axio-appendicular musculature suffers less than the appendicular muscles proper; not only are the muscles arising in the trunk and passing to the extremities less affected by the paralysis than the intrinsic muscles of the leg and arm, but the former give the first evidence of returning function. It may naturally be expected that voluntary movements will first return to some degree in the shoulder girdle and will be last to make their appearance in the intrinsic muscles of the hand. The same statement applies to the lower extremity, where movements at the hip-joint are first to return while those in the foot are late in reappearing if they do so at all.

As soon as any voluntary movements are observed in the muscles of the shoulder girdle their character should be noted and the patient instructed to repeat them frequently. All instructions concerning exercise should be made as specific as possible. A routine schedule will prove most efficient in attaining this end. Such a schedule should indicate the muscles to be exercised, the character of the exercise, the number of times the particular movements are to be performed at each

session as well as the number of sessions during the day. It is futile to begin a course of exercises unless some such systematic method is adopted.

The following exercises have been arranged with special reference to the muscle groups of the arm and leg. All of them may not be necessary in any given case. With the precise determination of the defective muscle groups it will be possible to select from this list the exercises demanded by a particular individual.

GROUP I.—AXIO-APPENDICULAR MUSCULATURE OF THE SHOULDER GIRDLE.—*Exercise 1.*—For the trapezius muscle, producing elevation of the shoulder. Disability of this muscle is observed in the drooping shoulder with loss of elevating power.

With the head immobilized, elevate the shoulder as in shrugging. The movement should be performed on both sides, since the associated movement of the unaffected muscle assists materially in the correct performance of the act on the paralyzed side. This procedure should be followed in all exercises until the defective movements show improvement. Associated exercises may then be discontinued. The elevation of the shoulder should be practised three times a day and repeated ten to twelve times at each session. This number may be increased as time goes on. It is well to begin all of the exercises for the shoulder muscles at the end of the second week, while the patient is still in bed.

Exercise 2.—For the pectoralis major muscle, producing ventral adduction of the humerus. Disability of this muscle is evidenced by the abducted position of the humerus from the body and the loss of the power of ventral adduction.

With the forearm flexed at right angles to the humerus the latter should be drawn inward across the chest. If the antagonist groups are paralyzed the arm may be passively abducted by the attendant and adduction in this way repeated the required number of times.

All of the movements for the muscles of the shoulder-joint are best performed with the patient resting upon the unparalyzed side.

Exercise 3.—For the latissimus dorsi muscle, producing dorsal adduction of the humerus. Disability of this muscle is evidenced by the abducted position of the arm and the loss of power in dorsal adduction.

With the forearm flexed at right angles to the humerus the latter should be drawn inward across the back. If the antagonist groups are paralyzed the attendant may passively abduct the arm and thus permit of repeated adductions.

Exercise 4.—For the deltoid muscle producing abduction of the humerus accompanied by elevation to right angles with the body. Disability of this muscle is evidenced by a flattening of the shoulder and loss of power to abduct the humerus.

With the forearm flexed abduct the humerus from the body. In the absence of adductor power the arm will naturally gravitate to the side.

Exercise 5.—For the deltoid, latissimus dorsi, and pectoralis major muscles.

Abduct and adduct the humerus from the body in several of the possible arcs through which abduction normal takes place, *i. e.*, in the ventral arcs or the dorsal arcs.

Exercise 6.—For the subscapularis, teres minor, and infraspinatus muscles, producing external and internal rotation of the humerus. Disability of these muscles is evidenced by the loss of rotatory power.

With the forearm extended rotate the humerus internally until the dorsum of the hand comes in contact with the thigh. Then reverse the direction and rotate the arm externally until the dorsum of the hand comes to lie upon the bed. When possible this exercise should be done against a slight amount of resistance offered by the attendant.

Exercise 7.—For the rhomboidei muscles, producing adduction of the scapulæ. Disability of these muscles is evidenced by a winging of the shoulder-blades and the loss of power to draw them together.

With the patient still resting on the unparalyzed side the scapulæ are alternately adducted and abducted.

Exercise 8.—For the combined action of the axio-appendicular muscles.

With the patient in the supine position the individual single muscular acts already practised may now be combined in a series of succession movements (diadochokinesis), beginning with a contraction of the trapezius and then followed in regular order by the deltoid, pectoralis major, deltoid again, latissimus dorsi, teres minor, infraspinatus, subscapularis, and rhomboidei. This will produce an elevation of the shoulder, abduction of the humerus, adduction of the humerus across the chest, abduction of the humerus, adduction again inward but toward the back, rotation of the arm, and finally adduction of the scapulæ. This series of diadochokinetic movements if consistently practised will reestablish all of the muscular combination possible about the shoulder-joint, and will thus lay the necessary foundation for the reintroduction of other impaired functions in the arm.

The means by which these exercises may be instituted will vary in different cases. It is often necessary at first for the patient to initiate the movements with the aid of the attendant and accomplish whatever little is possible for him. In many instances the patient may supplement the remnant of his former muscular power using his sound hand in assisting the paralyzed limb. This assistance should, however, be dispensed with as soon as possible and the patient encouraged to perform the voluntary acts alone. Movements against resistance at first but gradually increased will serve to strengthen the muscular contraction. This is easily done by the attendant who gently restrains the arm as the patient endeavors to move it into the several positions.

Fixing the attention upon the various movements is of great value. This is best accomplished by requiring the patient to watch the motions he is making with the shoulder and arm and from time to time comparing them with those of the sound side.

GROUP II.—MUSCLES PRODUCING MOVEMENTS IN THE FOREARM.—The muscles of this group are usually more profoundly affected than

those about the shoulder-joint. It is rarely the case that they give much evidence of returning function until the end of the second or third week. At about the time when the patient is to be gotten out of bed these muscles should receive attention. When the patient is able to sit in a wheel chair, the services of another helpful device may be enlisted. As already stated, much is gained by having the patient concentrate his thoughts upon the muscular acts he is performing. He should be instructed to observe closely all of his movements. If his chair is placed before a mirror he will not only be able to give his attention to each exercise but he may also compare the movements of the paralyzed arm with those of the unaffected limb which he is performing simultaneously. This also serves as a very valuable expedient in retaining and increasing the patient's interest.

Exercise 1.—For the biceps, producing flexion of the forearm.

With the elbows on the arms of the chair the forearm should be flexed to the full extent on the sound side and as much as possible on the affected side.

Exercise 2.—For the triceps muscles, producing extension of the forearm.

With the forearms resting on a smooth topped table placed in front of the patient the forearms should be extended as far and forcibly as possible. They should be held extended for several seconds before repeating the exercise. This is one of the most important of all the movements of the upper extremity, for it will be remembered that the tendency of spastic hemiplegia is the production of rigid contractures along the lines of greatest muscular traction, which in this case is that of flexion. Therefore extension of the forearm should be performed much more frequently than flexion and particular attention should be devoted to the action of the triceps.

Exercise 3.—For the pronator and supinator muscles. With the forearm resting on the table the hand should first be supinated and then pronated.

Exercise 4.—For the diadochokinetic movements of the forearm.

As soon as may be after the patient has acquired some degree of control over the separate acts already indicated it will be necessary to mould these into various series of succession movements. For this purpose it is well to adopt some definite serial act which may later on be modified in numerous ways. A good serial combination to begin with is the following: (a) Flexion of forearm; (b) extension of forearm; (c) pronation of forearm; (d) supination of forearm. When this combination has been fairly mastered, others, of which the same individual movements are the basis, should be tried.

In carrying on these movements all of the methods already recommended should be employed. Electricity may be used as a valuable accessory in this connection, especially in augmenting extension of the forearm. In using this auxiliary to the treatment the interrupted galvanic or sinusoidal galvano-faradic current may be used. A small electrode of sheet lead (6 cm. by 2 cm.) is firmly attached lengthwise

to the arm over the belly of the triceps muscle; a large electrode (15 cm. by 10 cm.) is placed at the back of the other arm. If the galvanic interrupted current is used the smaller electrode must represent the kathodal pole. The current is now passed through the arm at a slow rate of interruption and with each contraction of the muscle the patient is directed to forcibly extend the forearm. This purpose is even better accomplished by the use of the galvano-faradic sinusoidal current, since the interruptions are here less sudden and more rhythmical, thus affording the patient greater opportunity for exerting his will.

GROUP III.—MUSCLES PRODUCING MOVEMENTS AT THE WRIST.—The object here, even more than in the case of the forearm, is to combat the tendency of the flexors or overcome the extensors so that all exercises should be directed toward the development of these muscles.

Exercise 1.—With the flexor surface of the forearm resting upon the table extend the wrist first by the radial extensor, then by the ulnar extensor.

Exercise 2.—With the wrist held firmly upon the table by the unaffected hand, forcibly extend the wrist by using both sets of extensor muscles.

Electricity may be used with advantage to initiate or augment the extensor movements of the wrist in the manner already described.

GROUP IV.—MUSCLES PRODUCING MOVEMENTS IN THE FINGERS.—The fingers offer the greatest difficulty in the matter of exercise: (1) Because the intrinsic muscles of the hand as well as the digital flexors suffer more in the paralysis than any of the other muscle groups; and (2) because of the marked disparity between the flexor and extensors under normal conditions. The prevention of flexor contracture of the fingers is a troublesome problem, while the contracture once established in the fingers constitutes the most obdurate deformity which is met with in hemiplegia. Not a little may be accomplished by the use of the splints already recommended during the period of insult. If necessary they should be employed beyond this time. All efforts in the way of exercise must be directed to the several extensor groups of the fingers.

Exercise 1.—With the hand upon the cover of a fairly thick book, in such a manner that the fingers project over the edge, all of the fingers should be extended together. If the effort is feeble and the result unsatisfactory, assistance may be given by the sound hand and the extension thus performed a number of times. In this connection it may be stated that the patient will aid much in combating the tendency toward flexor contractures by grasping the paralyzed fingers in his sound hand and forcibly extending. This he should do a number of times each day.

Exercise 2.—With the hand placed as in Exercise 1, the patient should extend each finger separately and in succession. The use of electricity will be of much assistance in this exercise. A small examining electrode is best employed for this purpose, for by this means each muscle bundle of the extensor group may be stimulated.

Exercise 3.—With the hand in the position of Exercise 1, the fingers should be extended and spread apart by contraction of the interosseous muscles.

Exercise 4.—With fingers so placed on the table as to allow the thumb to extend over the edge, the adductor and extensor of the thumb may be exercised in the usual manner.

After a time the patient will have made sufficient progress to warrant the performance of more highly synthetized acts than those indicated in the exercises. Sustained voluntary processes of a simple character may be carried out with some degree of accuracy. Certain special exercises for this purpose have proved useful. Among these the first to be attempted is the management of alphabet or building blocks by the paralyzed hand. Later, marbles of several sizes may be put in a box and the patient directed to transfer them to some other receptacle. Turning the leaves of a book, shuffling and dealing cards, drawing lines with a pencil, using a planchet, and many other simple acts serve well to call into action the various muscle groups rendered defective by the paralysis.

As is the case in the arm, the axio-appendicular musculature of the leg is least affected and quickest to recover from such involvement as it may have sustained. It is a matter of general experience that the paralyzed leg makes better progress toward recovery than the arm. Exercises for the leg muscles should be commenced while the patient is still in bed. The chief object during this period is to overcome the tendency toward external rotation of the thigh by exercising the internal rotators particularly the gluteus medius and minimus. Other exercises for flexing and extending the leg on the thigh should be used.

LOCOMOTION.—It is not, however, until the patient is out of bed and able to stand that the real effectual work in reëducation of the leg begins. At this time the patient should have set before him in a simple way the essential mechanics of locomotion. The following points should be made clear to him so that he may intelligently set about the reconstruction or correction of defective components in his gait.

1. The biped gait presents two distinctly different phases.

(a) The phase of support in which the full weight of the body is sustained upon one leg.

(b) The phase of progression in which one leg is swung forward, backward, or to the side, as the case may be, in the process of taking a step.

Each leg alternates with the other in presenting these two phases.

2. The relations of the legs to the body and to each other during these phases. The leg in the phase of support is in line with the body and is practically locked firmly to the body at the hip-joint as well as rendered rigid at the knee and to a less degree at the ankle. The leg in the phase of progression rapidly changes its relations, for beginning at an angle to the body posteriorly it is swung through an arc that brings it to rest at a similar angle anteriorly.

The two legs at the beginning of the phase of progression form, with the ground, a right-angled triangle; the ground is the base of this triangle, the leg of support is the altitude, and the leg of progression is the hypotenuse. The length of the hypotenuse is greater than that of the altitude by reason of the fact that there is a greater amount of extension in the leg of progression, due principally to extension of the foot.

3. The length of the hypotenuse must be reduced to less than that of the altitude in order that the leg of progression may be swung forward or backward. This reduction in the length of the leg is accomplished by flexion; this flexion, however, is produced at the least possible expense to the individual muscle groups by the fact that the shortening results from a slight degree of bending at several joints instead of a large amount in any one joint.

The shortening of the leg takes place at four chief points:

1. By tilting or flexion of the pelvis.
2. By flexion of the thigh or the pelvis at the hip-joint.
3. By flexion of leg on the thigh at the knee-joint.
4. By flexion of the foot on the leg at the ankle-joint.

READJUSTMENT OF GAIT.—In the readjustment of his gait, therefore, the hemiplegic has two principal acts to learn, *i. e.*, locking of his leg of support in such a relation to his body that it may properly become the leg of progression and finally the shortening of the leg in this latter phase in such a way that it may be swung free of the ground and enter correctly into the phase of support.

As soon as it is evident that the patient has sufficient strength to stand he should be lifted to his feet and, supported either by two attendants or by the backs of two chairs, be directed to support his weight on his unparalyzed leg. He should then attempt to shift his weight to the paralyzed leg. Care must be had in this exercise lest the patient's strength has been overestimated. If it is impossible to support all of his weight on the paralyzed leg some portion of it may be borne and gradually increased as the exercise is repeated from day to day. It is essential that the position of the paralyzed leg while performing this exercise shall be correct. The external rotation of the thigh must be overcome, and to prevent this occurrence it will prove advantageous if the foot of the affected leg is placed on the floor by the attendant so that the toe points directly forward. With the patient in this position the following exercises may be carried out.

Exercise 1.—For the elevation of the pelvis, to aid in shortening the leg of progression.

With the patient standing on the sound leg and supported by two chairs the pelvis of the affected side should be elevated and depressed a given number of times.

Exercise 2.—For swinging the leg through the arc necessary to progression.

With patient in position of Exercise 1, the leg is swung like a pendulum backward and forward.

Exercise 3.—For flexion of the thigh on the pelvis.

With the patient in the position of Exercise 1, the thigh is flexed to the greatest degree possible.

Exercise 4.—For the internal rotation of the thigh.

With the patient in the position of Exercise 1, the entire leg is rotated internally so that the toe points inward.

Exercise 5.—For extension and flexion of the leg on the thigh.

With the patient in the sitting posture the leg may be extended and flexed. In this exercise assistance of the attendant will be necessary at first. This is also true of Exercise 6.

Exercise 6.—For the flexion and extension of the foot in the leg. This is best accomplished with the patient in the recumbent or sitting posture.

After the patient has to some extent regained the powers of locomotion it becomes necessary to direct the particular elements in his gait by special means. As soon as the patient is able to support the weight of his body on the paralyzed leg he should be instructed to practice standing in this position without the aid of the sound leg. At first this will be difficult because of the lack of confidence on the part of the patient, but by constant repetition, however, it will be possible for him to stand several seconds at a time on the paralyzed leg. This exercise is one of the fundamental necessities in correcting the disturbances of the gait. The importance of the element of time in the gait should be impressed upon the patient. He should be shown the advantage of supporting the weight of the body on the leg of support for the longest time possible, and equally the disadvantages of a shortened period of support. It is the tendency of these patients to reduce the phase of support upon the affected side to a minimum with a consequent shortening of the phase of progression. If, on the other hand, the phase of support in the paralyzed leg is lengthened to the maximum the unaffected leg may be swung through a normally long arc and the gait will thus present a stride approaching the average length observed in healthy individuals. This matter of the length of the stride is an important one, for many hemiplegics who have not had these particulars called to their attention fall into the habit of allowing about one-half of their locomotory functions to remain unemployed. Not only must constant attention be given to increasing the length of the stride but even more is needed in correcting the abnormal positions occasioned by the flexors and external rotators of the leg. To this end the patient must be directed to swing the paralyzed limb forward at each stride in such a way that the toe will tend to point inward. It will require much time to gain this control; indeed, it may never be entirely gained. In any event the intention to "toe-in" should be constantly in the patient's mind while he is walking, and however imperfect his attempts in this direction may prove practice should be consistently kept up. At first each step taken must be studied in detail and the effort made to shorten the leg of progression in the proper way. The majority of hemiplegics depend upon the elevation of the pelvis for this shortening. It may be necessary to make use of this

pelvic movement in the early stages of training the patient to walk, but the other points of shortening should not be neglected. Where possible the exercises should be carried on in a room with a smooth wooden floor. Under these conditions the following device will prove of assistance.

Three strips of half-inch moulding are laid parallel along the floor eight inches apart for the entire length of the room. At intervals of every thirty inches cross strips of the same thickness are made to intersect the three parallel strips at an angle of 45 degrees. In this way a number of compartments will be formed. The patient should then be instructed to stand with his foot in the centre of the first compartment. The toe of the paralyzed side should be so directed as to point toward the acute angle formed by the long middle strip and the first cross bar. As the patient begins to walk it will be necessary for him to clear the cross bars in succession and at the same time keep the paralyzed foot inside of the outer longitudinal strip. It will be seen that this contrivance compels the patient to shorten the leg so that he may clear the cross bars. In order to keep his foot inside of the outer parallel strip he must rotate the leg inward. Both of these acts are the particular desideratum of the exercises. The way in which the strips are laid out makes it possible to use the device when walking in either direction. As the patient acquires proficiency in locomotion the height of the cross bars may be increased and thus compel a greater amount of shortening in the leg.

Another simple apparatus consists of two inclined planes leading up to a small landing. If the paralysis is in the right side the right side of one inclined plane should be fitted with a series of steps 4 inches high and 30 inches apart. This is to be used as the ascending plane. The descending plane should be equipped with similar steps on the right side except that they must be one-half as broad. If the paralysis is on the left side the steps should be placed to the left of the inclined planes. A hand railing may be erected on either side of the apparatus. In using this device the patient is instructed to walk up the ascending inclined plane in such a way as to keep his paralyzed foot on the steps. This exercise will require a marked flexion of the paralyzed leg and thus favor the return of the impaired motor function. The steps of the descending plane serve to support the heel of the patient as he makes his way back to the floor. In this way he is forced to employ the anterior tibial muscles, which are important factors in the process of shortening the leg before it is swung forward at each step.

CEREBRAL EMBOLISM

Nature and Mechanism.—Embolicism of the cerebral vessels is usually of the arterial type. If functional disturbances result from such a condition it is generally due to the occlusion of a large arterial stem or one of its more important branches. The symptoms

produced in all cases depend, first, upon the immediate effects of sudden occlusion of a large or important arterial trunk which occasions profound alterations in the hydrodynamic condition of the brain, and secondly, upon the relatively late developments due to infarction and encephalomalacia. The damage wrought by emboli has been attributed to a special type of arterial supply which is found in many places in the body. The peculiarity of this blood supply depends upon the fact that the arterial vessels in these parts are the so-called terminal or end-arteries of Cohnheim. Such arteries are usually described as occurring in the kidney, brain, spleen, spinal cord, lung, heart, retina, and the region supplied by the superior mesenteric artery. In all of these localities infarcts are of common occurrence as a result of embolism. The anatomical explanation of this condition was said to depend upon the presence of the end-arteries in these parts with the consequence that contiguous areas in the same organ has little or no collateral arterial supply. End-arteries in the strict sense are not a *sine qua non* for infarction, and, as Adami states, "it is not the absence of anastomoses or of other arteries supplying the same area that is essential to infarct formation, but the absence of arterial anastomoses sufficiently large to insure the proper nutrition of a part, once the main vessel to that part becomes suddenly occluded."

According to Welch, emboli causing demonstrable symptoms are most frequent in the arteries of the kidney, spleen, and brain, the frequency of occurrence being in the order mentioned. These emboli are usually detached cardiac thrombi from the left side of the heart or from the heart valves. They may, however, be portions of aortic thrombi or necrotic thrombi from the pulmonary veins. Atheromatous material from the venous system may, in certain cases, produce arterial occlusion. This is known as paradoxical or crossed embolism and depends upon the patency of the foramen ovale in adult life. The communication between the two auricles of the heart has been found to occur in one form or another in 33 per cent. of cases. There is a well-known clinical dictum to the effect that all cerebral emboli spring from the left heart or the aorta and that the diagnosis of cerebral embolism must therefore depend upon the demonstration of a cardiac or aortic lesion. This undoubtedly is an excellent rule of thumb, but it must also be remembered that arterial emboli may arise from the pulmonary veins and that the persistence of the foramen ovale in a large percentage of cases affords opportunity for detached portions of venous thrombi to cause arterial occlusion.

Formation of Emboli.—Emboli may be formed in various ways as a consequence of pathological changes in the bloodvessels or the heart; they, however, may be extravascular in their origin and secondarily gain entrance into the blood stream. Not a little light is thrown upon the subject of cerebral embolism by the differences in constitution presented by the various kinds of emboli. The commonest cause of emboli is the detachment of thrombotic material from the walls of the vessels or chambers of the heart. The original thrombus may be of

several different varieties. Emboli due to atheromatous or calcareous material from atheromatous ulcers are next in point of frequency. Exfoliated cell-masses from tumors may enter the blood stream from emboli and, continuing their growth, give rise to metastatic neoplasms.

Exfoliated tissue cells, particularly of the placenta or the liver, may act as emboli. Fatty masses form another variety of the extravascular emboli. They almost never occur except as the result of trauma, operative or accidental. The fracture of the shaft of a long bone, liberating marrow fat, is usually the cause assigned to fat embolism. It may also be due to osteotomy, as well as to resection and forcible breaking down of ankylosed joints. The operative manipulations incident to the management of large pannicular adiposities, the handling of fatty omenta and mesenterics, or other fat accumulations, may likewise lead to fat embolism.

Certain gases may form emboli, as for example nitrogen. This condition is frequently seen among divers and compressed-air workers in general. Nitrogen embolism, although the most frequent, is not the only form of gas embolism. Thus Janeway and Hun report a case with severe cerebral symptoms due to the injection of hydrogen peroxide into the abdominal and thoracic cavities. Certain gas-producing bacteria may cause the same effect, especially the *bacillus aërogenes capsulatus* of Welch.

Some animal parasites having gained entrance into the circulation are capable of acting as emboli. Collections of the trypanosomes in the cerebral capillaries are said to cause the symptoms of the sleeping sickness. Rupture of an echinococcus cyst into a vein may also give rise to embolism. Intravascular emboli in consequence of disease of the heart or bloodvessels, are undoubtedly of the most frequent occurrence. The other varieties, however, should not be lost sight of as bearing upon the possibilities of diagnosis and treatment. As already stated the effects of cerebral embolism are generally of two types in any given case—those which are immediate and those which are relatively late. The late symptoms are focal in character, while the immediate symptoms are more generalized. The question has often been raised why the occlusion of a single vessel in the brain should produce such profound cerebral disturbance. In many respects the solutions offered to this problem are still somewhat unsatisfactory, but Geigel's explanation, given as his view of the mechanism of the apoplectic insult in embolism, seems to be the most logical and best founded on physical grounds. In Geigel's opinion the instant that an embolus becomes effective in one of the larger cerebral arteries a great circulatory change takes place in the entire brain. All of the arteries which are not occluded become dilated and tend further to express the blood from the embolized area, with an attendant engorgement of all other regions. In this way an acute nutritional disturbance is determined in the brain which has been termed *Adiä-morrhysis*. Some time is required for the reëstablishment of circulatory equilibrium after the insult and during this interval the disorder is characterized by generalized cerebral symp-

toms, the patient usually being comatose. When the adjustment has been accomplished, however, and the graver symptoms pass, the patient then gives evidence of extensive focal lesions in the brain which are usually manifested by hemiplegia. If, on the other hand, relatively small arteries of the cerebral vascular system are occluded, the more profound general symptoms may not appear, and in this event only the phenomena due to the focal disturbance will be observed.

Here, as in the case of cerebral hemorrhage, there are three stages of the disease, each requiring a more or less distinct course of treatment.

Treatment of Period of Preparation.—Some of the causes operative in the preparation of cerebral hemorrhage are the forerunners of cerebral embolism. This is true of arteriosclerosis, especially as it has to do with atheroma and calcification of the bloodvessels. There are, however, numerous other causes paving the way to cerebral embolism and the general principle which underlies them all is not so much a weakening of the arterial walls as it is the introduction into the circulation of foreign bodies either as a result of disease of the cardiovascular lining or from some extraneous source. Another important element is found in the various changed conditions of the blood itself, changes which involve its viscosity or coagulability or bath. The most prolific causes of embolism are diseases of the cardiovascular intima and such alterations in the character of the blood as lead to thrombosis. The first step in the treatment directed against the occurrence of cerebral embolism should hold these points in the foreground.

Every case of acute endocarditis may give rise to cerebral emboli, although the first attacks even of the more severe forms are less often responsible for the complication than the recurrent variety of endocardial inflammation. It has been estimated that 89 per cent. of cerebral embolism may be traced to some cardiac disease. Endarteritis, especially involving the ascended and arched aorta, is also a frequent source of embolism. Contrary to the somewhat general opinion the endocarditis occurring as a complication of certain febrile diseases should be regarded as having its menace in the formation of cerebral emboli. Such a complication in the course of pneumonia, diphtheria, puerperal and general septicemia, typhoid fever, and other exanthemata is fraught with the dangers of embolism. Endocarditis occurring independently of such diseases should be thoroughly investigated; blood cultures studied, and autogenous vaccines prepared. Stock polyvalent vaccines of the staphylococcus and streptococcus often give excellent results. It is the experience of the writer that endocardial inflammation due to the pneumococcus responds most satisfactorily to autogenous vaccines. Vaccination should always be considered in the case of endocarditis occurring intercurrently with some other acute infectious disease. The blood pressure should be made a matter of careful study in these cases, for embolism is usually accompanied by relatively high blood pressure. If the effort of rising, sitting up or standing causes any noticeable increase in blood pressure, all activity must be interdicted, the patient confined to the bed, and kept as free from

excitement as possible. Jones' figures show that 55 per cent. of all cerebral embolism occurs while the patient is in bed. It is probable that the number of cases upon which these figures were computed were not large enough to permit the determination of an adequate average.

Mechanically it would seem obvious that whatever causes tended to produce variations in the blood pressure, favoring its elevation, must be avoided. Recurrent and chronic endocarditis are more prolific causes of cerebral embolism than the acute variety. To a less degree arteriosclerosis with its atheromatous and calcareous degenerations of the vessels is the essential disturbance in many cases. The treatment in such cases must be directed to the regulation of the habits and general life of the patient. Violent exercise, unusual exertion as well as mental stress should be prohibited. The patient must be instructed to live within his means, as these are represented by the condition of his heart and bloodvessels. The iodide of potassium is undoubtedly the most useful agent in treating these conditions.

Thrombosis is another cause of embolism, but its general character is so little understood that the lines of treatment are still limited. Whether the condition is due to a disease of the blood causing a change in its coagulability or viscosity, or whether it is the result of disease in the vessel, has not yet been satisfactorily made out, in consequence our therapy in this instance is still tentative and empirical. Potassium iodide has been highly recommended. The various manners and dosages in which it may be administered have all found advocates.

For the prevention of embolism arising from the more common extraneous emboli, emphasis should be laid upon the necessity of instructing compressed-air workers in the proper method of slowly decompressing air workers in the proper method of slowly decompressing themselves before returning to atmospheric pressure. In this way the percentage of nitrogen embolism will be greatly reduced. Fat emboli may be thrown into the circulation as a result of trauma, operative or accidental, to fatty tissues. Nothing may prevent the occurrence of fat embolism following fractures, on the other hand care should be taken in all operative procedures to handle the large masses of fatty tissue with the least possible amount of manipulation.

Treatment of Period of Insult and Period of Paralysis.—The essential differences between cerebral hemorrhage and embolism are confined to the period of preparation. In the period of insult it is often impossible to distinguish between these two conditions, in which event the general directions already stated for the management of the period of insult in cerebral hemorrhage should be followed. Often the blood pressure is high in this phase of embolism, a symptom which at first sight suggests hemorrhage. Much reliance has formerly been placed in the finding of a cardiac lesion as evidenced by a heart murmur. This in itself should not be accepted as anything more than suggestive, since cardiac lesions often complicate cerebral hemorrhage, while venous thrombosis is one of the most frequent causes for cerebral

embolism. It may be set down as a general rule that all apoplectic cases in the period of insult having a high arterial tension should receive treatment which is especially directed against the elevated pressure.

The proposition to the effect that increased blood pressure in these cases is salutary, cannot be seriously considered. If this high pressure has as its purpose the maintenance of the hemic equilibrium in the vital centres during the insult why do these patients so uniformly present high blood pressure both before and after this period? Furthermore, what conservative element may accrue from a sustained high pressure in a vascular system designed by nature to transmit a fluid at relatively low tension. The immediate treatment of embolism should therefore be similar to that of hemorrhage. The details of accommodating the patient, of removing his clothes, of his position in bed, of the medication for attempting to reduce the blood pressure and transportation if necessary, have already been discussed in the treatment of the period of insult in cerebral hemorrhage. The same may be said of the period of paralysis except that further restrictions must be put upon the patient on account of his cardiac or arterial disease.

CEREBRAL THROMBOSIS

Nature of Mechanism.—In taking up the subject of cerebral thrombosis we enter a field at once complex and promising, for although the nature of this condition gives rise to some intricate questions in biochemistry, the fact that the fundamental character of the disturbance is so evidently chemical offers much hope for the ultimate solution of the problem.

By thrombosis is meant the clotting of blood inside of the cardiovascular system during life in contradistinction to coagulation which is blood-clotting outside of the vessels. A thrombus, therefore, is a clot within a vessel or the heart, consisting of fibrin, a white, insoluble protein precipitated in the form of a network of fine fibrils. This precipitation occurs as the result of certain chemical changes in the blood and the reduction of fibrinogen, a substance normally present in the blood and lymph, to an insoluble, fibrinous state.

Conditions of Thrombosis.—Clotting in the vessels only occurs under definite conditions which depend upon three elements: (1) Thrombogen; (2) lime salts; (3) an enzyme or ferment. Thrombogen and the lime salts are normally present in the blood, but the ferment appears there only under unusual circumstances. It would seem that this last mentioned element is a *tertium quid* to the formation of thrombus, and that inasmuch as its presence in the blood is unusual, its nature should be all the more carefully inquired into. This ferment is found normally in a number of places in the body, as, for instance, in the muscles, walls of bloodvessels, glands, erythrocytes, and leukocytes. It has been termed thrombokinase by Morawitz and cyto-zym by Fuld and Spiro. Coagulation and thrombosis, therefore, represent the

reaction between fibrinogen and thrombin, but thrombin does not exist in the blood as such, it is present as an inactive substance known as prothrombin or thrombogen. This latter substance, under the influence of the ferment thrombokinase, may be reduced to thrombin, and then, in the presence of lime salts, coagulation occurs, erythrocytes and leukocytes. Under normal conditions this ferment has its nearest approach to the blood stream in the walls of the vessels, from which, however, it is separated by the endothelium. Any injury or disease to this endothelium would open the way for the entrance of thrombokinase into the blood and thus favor formation of a thrombus. On the other hand, this same outcome may eventuate without primary disease or injury in the endothelium of the vessels, since any cause determining the destruction of red and white cells may liberate thrombokinase and so lead to thrombosis. In general, then, it may be stated that there are three possibilities leading to the formation of thrombi: (1) A disease or injury to the blood-vascular system in any of its parts causing a change or destruction of its endothelium; (2) pathological conditions producing changes in the blood itself; (3) both of these two factors acting simultaneously.

Very interesting in this connection and at the same time shedding light upon the ferment theory of coagulation are the observations of Delezenne and Nolf. These investigators have shown that the blood of fishes, reptiles, and birds coagulates very slowly when carefully removed from the bloodvessels and drawn off into glass tubes. If, however, the blood in its escape is permitted to come in contact with the surface of the wound through which it is being drawn coagulation rapidly takes place. This reaction, it is claimed, indicates at once the presence in the tissue of a blood-clotting ferment and demonstrates the defensive means with which nature has provided the organism to prevent extensive or fatal hemorrhage. The cell-free blood plasma of the animals above mentioned if rapidly centrifugated coagulates slowly or not at all, but if tissue extracts are added then coagulation is rapid and marked.

Howell's View of Thrombosis.—In more recent times another view of coagulation and thrombosis has been advanced. This is the view of Howell; it is based on a large number of experiments. Its premises are lucid and definite, its conclusions logically and forcibly drawn. If this new theory proves to be entirely in accord with all the facts a long step forward has been taken not only in the understanding of the mechanism of thrombosis, but equally as much in the treatment of the conditions which cause it.

According to Howell the blood contains an inhibiting substance which prevents the activation of prothrombin by the calcium salts, with the resulting formation of thrombin. It will be remembered that coagulation requires the presence of thrombin and fibrinogen together, so that this inhibiting substance, precluding as it does the formation of thrombin, makes coagulation impossible. The inhibiting substance Howell calls antithrombin. If, however, this antithrombin becomes

neutralized in some way coagulation occurs promptly. It is generally known that there is a substance in blood plasma and the so-called peptone plasma which has the power of inhibiting the action of thrombin upon fibrinogen with the production of a clot. The following experiment of Howell indicates this clearly:

To 5 c.c. of fibrinogen was added 5 drops of a thrombin solution which yielded a firm clot in five minutes. Then using the same amounts of fibrinogen and thrombin a few drops of peptone plasma heated to 60° C. and filtered was added, with the result that no coagulation occurred in twenty-four hours. The peptone plasma therefore appears to contain a substance which inhibits the action of thrombin on fibrinogen. This substance so closely resembles antithrombin in its general properties that it is fair to call it by this name. Commercially this substance is known as hirudin. It is a principle obtained from the heads of leeches by physiological salt solution, used to prevent coagulation of the blood and may be employed in doses of $\frac{1}{80}$ grain (0.001 gram). While this experiment demonstrates the presence and action of antithrombin in the blood plasma it next became necessary to establish the existence of an element which neutralized antithrombin. To this end a mixture of fibrinogen thrombin and peptone plasma was made with the usual result of no clot formation. To this a tissue extract from the spleen and thymus was added and immediately coagulation occurred, thus showing that the tissue extract contained as it were an antibody to antithrombin. This antibody Howell calls thromboplastin.

Thromboplastin occurs in many tissues of the body into which the blood escaping during hemorrhage would rapidly have its contained antithrombin neutralized with the consequent formation of a clot. Clot formation in these instances is a conservative process, and because almost every tissue in the body seems to contain thromboplastin the entire economy is provided with a substance capable within limited bounds of meeting the emergency of hemorrhage. When clotting takes place within the cardiovascular system thromboplastin has gained entrance into the blood from some source, either because the corpuscular elements themselves are undergoing pathological changes or because of certain diseases in the walls of the bloodvessels. Howell's theory not only offers a new field for investigation but brings with it the hope that the conditions leading to thrombosis may be successfully combated.

Conditions Favoring Thrombosis.—Certain conditions distinctly favor the formation of thrombi, thus slowing of the blood current with an attendant reduction of pressure, according to Lubarsch, is especially favorable to thrombosis. Thrombi occur four times as frequently in the veins as in the arteries; they occur when the vascular channels are unusually wide or dilated, as in the auricular appendages in the sinuses of the brain, and in certain aneurysms. If the blood current is rapid and under relatively high pressure the tendency to thrombus formation is slight; and this is so even in spite of injury or disease to the endothelium of the channel, a fact well illustrated by the infre-

quency of thrombosis of the aorta even in the presence of atheromatous ulceration there.

The formation of eddies in the blood current has been held responsible for thrombus formation. According to this idea the blood at the centre of the eddy is moving more slowly, and hence has greater opportunity to clot.

Hemolysis and destruction of the blood corpuscles also seems to play a part in thrombosis. This destruction occurs as a result of certain endogenous and exogenous agents. Among the exogenous poisons which may act in this way are the salts of lead, mercury and arsenic, the sulphates, chlorates and sulphates of potassium, toluylenediamin, nitrobenzole, phenalhydrazin, carbolic and salicylic acids, extracts of amanita and other poisonous mushrooms, snake venom, and many enzymes. Common among the endogenous poisons are the products of extensive burns, toxic substances of uremia, severe secondary anemia, as that accompanying carcinoma and pernicious anemia.

Compared with bacterial toxemias, those due to endogenous and exogenous poisons are relatively rare. According to Adami most people die of terminal infections while the so-called bland thrombi observed in these cases are found to contain bacteria. Welch and Lubarsch maintain that if known cases of suppuration, lobar pneumonia, typhoid fever, appendicitis, diphtheria, acute rheumatism, measles, and influenza be carefully studied, capillary thrombi in the brain, lungs, kidneys, and intestines will be found to occur with remarkable frequency.

Another predisposing cause of thrombosis, and perhaps the most prolific one, is disease or injury to the vascular walls. In the opinion of some pathologists this is the condition essential to thrombosis. Experiments by many observers seem to indicate that impairment of the vascular endothelium is not in all cases a sufficient cause for thrombus formation. A roughened endothelial surface, however, over which the blood is passing at an unusually slow rate and at low pressure provides a combination of conditions which almost inevitably produce thrombi. The causes which may determine the angitis are numerous and varied. They comprise the factors which have already been mentioned in connection with thrombosis due to changes in the blood. So that it does not overreach the facts to state that the great majority of diseases may pave the way to thrombosis and that, in this light, many of the sequelæ of such diseases perhaps have their explanation, at least in part, in the formation of thrombi. It would seem that this proposition opens a wide field for thought and investigation inasmuch as there is now the likelihood that the tendency toward thrombosis may be overcome therapeutically.

Treatment in the Period of Preparation.—If such a period may be recognized at all it will, as previously stated, extend over a wide range of diseased conditions; it will introduce a new object for care and attention during the defervescence of and recuperation from long febrile illnesses; it will render necessary the routine investigation

concerning the clotability of the blood in cases of cachexia and anemia and in all such cases it will raise the question as to the expediency of attempting to control the coagulability of the blood by the therapeutic means which seem now to be at hand. It may be impossible to avoid or eliminate the agents which do damage to the vascular endothelium and thus favor thrombosis, but something may be accomplished in overcoming the tendency to a slow-moving, low-pressure blood current and even, perhaps, to combat an increased disposition on the part of the blood to clot.

Blood pressure below 100 mm. in the adult should always be looked upon with suspicion. This is especially true if the pulse is irregular or arrhythmic. Whatever the causes of this lagging circulation may be, whether exhaustion, failing compensation, cachexia, or anemia, the condition should not be allowed to continue.

Therapeutic Indications.—Under such circumstances the therapeutic indications are for such drugs as increase the force of the heart and at the same time decrease its frequency. For this purpose digitalis, strophanthus, scilla, convallaria, and scoparius may be used. Digitalis gives the most uniformly satisfactory results. Care must be had to disturb the digestion as little as possible. The tincture of the drug is, perhaps, the best adapted to administration by mouth. The dosage must be suited to the requirements of the individual case. It may become necessary to employ some of the other agents mentioned above or a combination of several of them. A useful combination of this character is the following.

R—Tincturæ digitalis	m̄v
Tincturæ strophanthi	m̄ij
Elixiris aromatici	3j

This may be given every three hours according to the nature of the case. Another good combination is found in the Waldstein tablet, which contains the following:

R—Sparteïn sulphate	gr. $\frac{1}{10}$
Tincturæ strophanthi	m̄ij
Caffeine citrate	gr. $\frac{1}{4}$
Codeine sulphate	gr. $\frac{1}{10}$

Adrenalin hydrochloride and strychnine sulphate have also been recommended for the same purpose.

In determining the clotability of the blood the following simple apparatus of Dale and Laidlaw may be employed.

1. Capillary tube 1.3 to 1.4 mm. in diameter, 2 cm. long.
2. Small shot large enough to roll through the lumen of the tube.
3. A basin for holding water at a temperature of 35° to 40° C.

A drop of blood from the finger is introduced into the tube and the ends of the tube sealed with plasticine. The tube is now held under warm water and tipped up and down allowing the shot to roll from one end to the other. The instant the shot stops rolling is recorded by

a stop-watch. The time elapsed between the drawing off of the blood and its clotting in the capillary tube is known as the coagulation time. According to this method of estimation the average coagulation time in the healthy individual is one minute and forty-five seconds. A coagulation time of one minute or under should be considered as abnormally rapid. It is premature as yet to recommend the use of anti-thrombin in the effort to inhibit or prevent thrombosis. Clinical evidence bearing upon these points is still insufficient. There are good reasons for the belief, however, that some means of introducing anti-thrombin without the dangers of anaphylaxis or other toxemias will soon be forthcoming.

Certain dietetic precautions have been advised to prevent thrombosis, especially after typhoid fever or other exhausting diseases. Partial decalcification of the milk by adding 0.25 per cent. or 0.5 per cent. of sodium citrate has been recommended. Wright and Paramore found that the coagulation time of the blood was reduced one-fifth by adding calcium chloride or lactate to the diet. This observation was not substantiated by Dale and Laidlaw.

Cases of low blood pressure, in which the patients complain of numbness, paresthesia, heaviness, or pain in one side of the body, or one side of the face, demand prompt and active treatment. Such conditions in conjunction with transitory vertigo or syncope, speech, visual, or auditory disturbance should be regarded as premonitory symptoms of cerebral thrombosis, for contrary to what was once the opinion concerning vascular disturbances of the brain, it is believed that cerebral thrombosis more frequently causes prodromal symptoms than hemorrhage or embolism. The blood pressure in such cases should be carefully followed and should be raised by the use of appropriate cardiac stimulants. It is often advisable to begin at once with some of the rapidly diffusible stimulants, as whisky or spirits of ether, and later introduce digitalis, sparteine, or strophanthus.

Treatment of the Period of Insult.—It is not often possible to differentiate at once between cerebral hemorrhage, embolism, and thrombosis. For this reason the immediate management of the patient should be the same in all cases. These points have been covered in discussing the treatment of the period of insult in cerebral hemorrhage (page 439). The first fact to be ascertained after establishing treatment is the height of the blood pressure. A low blood pressure in the absence of any cardiac murmur may be regarded as presumptive evidence of thrombus. An irregular tumultuous heart action with a rapid coagulation time of the blood strongly favors the diagnosis of thrombosis. Here, unlike the treatment of hemorrhage, the chief object is to steady and regulate the heart. If the blood pressure is low and the heart irregular or arrhythmic there is need of digitalis or strophanthus. If, on the other hand, the blood pressure is not below 90 mm. Hg. and the heart is regular it is very questionable whether any drugs of this type should be used. Another condition which contraindicates the use of digitalis or strophanthus is acute parenchymatous nephritis. With this com-

plication present the most reliable drug to use is sparteine sulphate, gr. $\frac{1}{4}$ every four hours. The patient should be kept flat on his back without elevation either of the head or the bed itself.

In other particulars the treatment of this period corresponds to that of hemorrhage and embolism as already described (page 439).

Treatment of the Period of Paralysis.—Particular attention should be given to cases of thrombosis in the period of paralysis in order to avoid a recurrence of the trouble. To this end the conditions of the circulation must be carefully watched; when the heart flags and the blood pressure is below 100 mm. Hg. proper cardiants should be employed. In thin, anemic individuals forced feeding and out-of-door life is to be recommended even to the extent of sleeping in the open air. Inasmuch as it is desirable to improve the cardiovascular tone of these patients they may be urged into more active exercise than in the cases of cerebral embolism or hemorrhage. The directions for these exercises and the methods of supplementing them have already been given on page 449. The patient should be especially encouraged in this part of the treatment, since excellent results are obtained in many cases.

CHAPTER XI

TREATMENT OF DISORDERS OF EXPRESSION (APHASIA, APRAXIA, ETC.)

By S. A. KINNIER WILSON, M.D.

It may be said at once, by way of introduction, that the subject of the treatment of disorders of expression is so large that it is desirable it should be delimited and the scope of this article defined. The following topics, then, will not be alluded to in this place:

1. Hysterical disorders of expression: hysterical aphonia, mutism, etc.
2. Deaf-mutism.
3. Other functional conditions: speech neuroses, such as spastic aphonia or aphthongia, stammering, stuttering, lisping, lalling, idio-glossia, etc. For a consideration of these speech defects the reader is referred to other chapters. In this article attention will be directed to three main disorders of expression, viz., aphasia, dysarthria or anarthria, and apraxia, and the treatment of these will be considered in turn.

It must be clearly understood at the outset that in any discussion on the therapeutic procedures to be adopted where such phenomena show themselves, we are dealing with symptoms, and symptoms only. There is no such disease as aphasia, just as there is no such disease as paraplegia. Similarly, apraxia, or aphasia of the extremities, as it has been somewhat inadequately termed, is merely a symptom, an exteriorization, of an underlying intracranial defect, and we shall lay ourselves open to the charge of irrationally attempting the impossible if we endeavor to "cure" a "symptom." Treatment must be directed, in the first place at least, to the underlying morbid condition, of which aphasia is but the outward expression. On the other hand, when the symptom seems to have become persistent it may be possible by suitable methods to alleviate or ameliorate or palliate its severity, and this constitutes a division of the subject to which considerable attention will be devoted.

It may further be remarked in this place that in the matter of aphasia and apraxia we are dealing with vexed questions. Round the subject of aphasia recent discussion has raged with an intensity that one had almost thought was foreign to any scientific study: and apraxia is a subject which is as yet comparatively little known to the profession at large, though of its importance and localizing significance there can be no doubt. For these and other reasons it appears appropriate to preface the consideration of the treatment of these conditions with some attempt at indicating what is the present state of our knowledge

so far as they are concerned; and in the case of apraxia a brief introduction on the matter of definition, terminology, and symptomatology is called for.

From the nature of the case it frequently happens that the symptoms of aphasia and apraxia clear up of their own accord; transient disturbances of this description are often seen in the clinical picture. This being so, the reader will appreciate the desirability of discussing prognosis and treatment together, and in the subsequent paragraphs it will be seen that this is the plan which the writer has adopted.

APHASIA

The intricacy of the subject of aphasia is attributable to two elements in its study; there is first the question of the regional diagnosis of the lesion producing the phenomena, and there is in the second place the question of the relation of these phenomena to the psychical and physiological functioning of the cerebrum. In other words, a given aphasic symptom-complex may be looked at in two ways: what is the site of the lesion producing the symptoms, and what is the nature of the physiological or psychical disturbance produced? Failure fully to appreciate the difference between these two questions—for they are on different planes—has frequently led to confusion of thought, and consequent misunderstanding. Every case of aphasia is thus double-sided, and this double-sidedness is reflected in its treatment, for that may be directed either to the improvement of the anatomical or rather pathological substratum, or of the psycho-physiological disturbance, and in each instance the fashion of the treatment differs widely from the other.

In the strict sense, of course, the two cannot be separated, and in dealing with the treatment of aphasia we must always bear in mind that, as Bastian says, words are the counters by means of which thought is carried on, so that *ipso facto* every case of aphasia is so far a case of impaired intelligence. But that, apart from the special defect of intelligence constituted by itself, aphasia may occur without any further intellectual defect of any sort, is abundantly clear from numerous clinical instances.

It is, fortunately, unnecessary to enter into the minutiae of the regional diagnosis of aphasia in this place. The writer assumes that the reader is familiar with the classical views which have been built up by the labors of half a century—for it is almost exactly fifty years since Broca made his epoch-making discovery—and suggests that he, further, need not feel that the accumulated evidence of these fifty years has suddenly become untrustworthy because of recent onslaughts on the classical theories. What cannot be shaken will remain, and the salient truths of the accepted aphasia teaching stand out the more clearly as a result.

Classification.—Aphasia is classified as motor or sensory. Roughly speaking, motor disturbances of expression are associated with cortical

or subcortical lesions situated in front of the fissure of Rolando, and of an imaginary line continued down from it across the temporal lobe; sensory disturbances of expression are linked to lesions situated posterior to the fissure of Rolando.

Motor Aphasia.—The motor speech centre is placed in the posterior portion of the third or inferior frontal gyrus of the left hemisphere, in right-handed people, where the gyrus borders on the operculum. Niessl von Mayendorf would place the centre in the lower end of the precentral gyrus, to the exclusion of the inferior frontal gyrus, but including the operculum; Pierre Marie would also dismiss Broca's convolution from its place of honor and locate the centre in a vast quadrilateral which includes both gray and white matter in profusion. Of the former view it may be said that while it does not differ so widely from the accepted theory as may appear, it does not commend itself for several reasons, which cannot here be entered on; of the latter there is no evidence of the existence of an actual speech "centre" in the quadrilateral, while it contains practically the whole of the projection fibers of Broca's convolution, as well as the associational fibers uniting the auditory word-centre (see below) to Broca's area; and it is already admitted, as Bastian and Collier say, that lesions of either of these paths is productive of aphasia by isolation of Broca's centre. A lesion of the motor speech centre is followed by inability to translate ideas into words; the patient is speechless; he may be able to "utter" an occasional word, under the stress of emotion or otherwise, but he cannot "speak," *i. e.*, he cannot "propositionize," and the words which he may get through, such as "yes" or "no," are usually incorrectly used from the point of view of ordered speech.

It is to be noted, however, that there is no paralysis of the muscles concerned; at least, in a pure case; so that while the patient cannot propositionize "yes" or "no," he can articulate the words perfectly well. We are thus led to the conception of inability to perform certain movements, *viz.*, those which are concerned in the formation of certain sounds which we call words, while at the same time there is no actual paralysis of the appropriate muscles. This phenomenon is denominated motor apraxia, or, more shortly, apraxia, and for the sake of synthesizing our conceptions there is every reason why pure motor aphasia should be considered as one, of several, varieties of apraxia. Broca's centre, then, may be viewed as a eupraxic centre for the orderly coördination of the successions of delicate movements necessary for the production of the spoken word; the word-images, which are in part at least kinesthetic images, must be awakened in this coördinated way prior to the actual innervation of the muscles concerned from the motor area proper, on the precentral cortex in front of the fissure of Rolando, at its lower extremity.

When the lesion involves the pyramidal fibers—as opposed to the subulcine fibers uniting Broca's eupraxic centre to the corresponding motor area *sensu strictiori*, or the eupraxic centre itself, or even other fiber groups reaching that centre from elsewhere (to be referred to

later)—which pass from the cortical motor centres for lips, tongue, palate, larynx, and so on, to the corresponding pontomedullary nuclei, by the *faisceau geniculé*, we have dysarthria in varying degrees of intensity to complete anarthria. Articulation is defective, apart from the content of the patient's speech. (It may be noted, parenthetically, that the use of the term anarthria by Pierre Marie differs materially from this the accepted usage.)

There is another motor centre of the higher, or eupraxis, type which is intimately associated with the carrying out of the expression of speech, in the wide sense of the word; this is the so-called writing centre, which is placed at the posterior end of the second or middle left frontal convolution. Written speech is an integral part of motor speech expression, but there has been much controversy as to whether a separate writing centre, in a narrow sense, should or could be postulated. If there have been observers, such as Dejerine, who have argued strongly against the existence of a distinct writing centre, there are others who have adduced clinical and pathological evidence in favor of it which it is difficult to gainsay. In a lesion of this area, the patient is unable to execute the ordered sequence of fine movements necessary to the production of letters and words with a pen, yet he need not be paralyzed at all as far as the arm and hand and fingers are concerned. Here again, evidently, we meet with the conception of a eupraxis centre for the building up in order of the succession of delicate movements concerned with writing, the actual innervation of the muscles taking place from their motor centres on the precentral gyrus. We thus come to look upon agraphia as a variety of apraxia, a view advocated by Liepmann, Heilbronner, and others in a convincing way. And there is little reason to doubt that by analogy some such eupraxis centre for the leg can be hypothecated at the posterior end of the first or superior frontal convolution.

It is worth noting that these eupraxis centres are situated in a region of the cortex which differs histologically from the motor area properly so-called, the excitable motor area, which corresponds to the anatomical region in which are found the giant cells of Betz. The eupraxis centres are placed in the intermediate precentral area, whose cortex differs also from that of the frontal pole, or rather, prefrontal region. It approximates more to the former than to the latter, however.

Sensory Aphasia.—Sensory aphasia is occasioned by a lesion which involves either the posterior end of the first and second temporal convolutions on the left side, or the cortical superficies in the region of the angular gyrus. The type is distinguished as auditory or visual aphasia respectively. In auditory aphasia the patient is unable to understand what is said to him, although he has no deafness; in visual aphasia he cannot understand what he reads, although he is not blind. The two varieties frequently occur together, and every gradation from slight to severe sensory aphasia is possible.

Now when a patient is unable to recognize what he sees or hears, he is suffering from a condition which is designated today as agnosia,

a condition which on the sensory side corresponds to apraxia on the motor side. By allowing such heterologous terms as mind-blindness, object-blindness, tactile aphasia, optic aphasia, and so on to fall into merited desuetude, we can adopt the expression agnosia to cover all varieties of failure to recognize stimuli reaching the sensorium by any sense avenue whatever, though the stimuli themselves are appreciated by the corresponding cortical region adapted to receive such stimuli. It will be seen at once, then, that auditory and visual aphasias are but varieties of agnosia, and the physician will do well to make himself familiar with this larger view of these disturbances.

Combinations of Motor and Sensory Aphasia.—Various combinations of motor and sensory aphasia occur, and, further, there are types of aphasic defect distinguished as transcortical, where Broca's and Wernicke's areas are cut off from a supposed higher cortical ideation centre. It is not, however, germane to the purpose of this article to enter into the refinements of aphasic diagnosis; apart from their comparative rarity, the interest of these types is chiefly academic, inasmuch as their pathology does not rest on an established foundation. The symptoms of transcortical aphasia occur only as the result of complicated cortical and subcortical lesions, and the treatment of the disease does not depend in any material degree on the complexity of its external manifestations.

Nothing further, therefore, need be said about the subdivisions of aphasia in this place, or on various anomalous symptoms occasionally met with in the study of cases.

Psycho-physiological Speech Mechanism.—Attention must now be directed to the consideration of certain problems which exercise an important bearing on the matter of prognosis and treatment in aphasia. As has already been hinted, a due appreciation of the double-sidedness of aphasia is necessary to enable the physician to gauge aright the nature of the morbid phenomena; the questions now to be discussed are concerned more particularly with the psycho-physiological side of the speech mechanism.

Function of Right Hemisphere.—What share does the right hemisphere take in speech? There can be little doubt that, as far as hearing, seeing, smelling, and tasting are concerned, the corresponding centres of both hemispheres are educated concurrently by suitable afferent stimuli. It has never been seriously maintained that hearing or vision preponderates in one as opposed to the other hemisphere. On the other hand, as Bastian points out, this is probably not the case for the tactile and other common sensibility centres, seeing that tactile and kinesthetic impressions have in all cases to be strictly localized. If, therefore, in the development of the child the right limbs are used more than the left, it is possible that the left hemisphere will preponderate over the right, will, in other words, become the leading or superior hemisphere, by reason of its reception of more afferent stimuli. Although no doubt there is in most people an innate or inherited superiority of the left hemisphere over the right, it is important to

recognize to what extent this is confirmed by education. The teaching of children to write with the right hand is universal, so that should a child not have by nature that superiority of the left half of the cerebrum over the right, to which reference has just been made, education will speedily determine it. There are many instances in the experience of everyone where a natural tendency to left-handedness has disappeared under the influence of training, especially, for some reason, in the case of girls, but the contrary process—a right-handed child becoming left-handed—never occurs. Indeed, it has been maintained by Ernst Weber that the epoch-making discovery of Broca in 1862 became possible, although right-handed men had existed for hundreds of years, only because the dissemination of the faculty of writing among all classes finally determined the superiority of the left hemisphere over the right, and at the same time finally established the localization of the speech centres in the left cerebral cortex. This position is perhaps rather too extreme, but enough has been said to indicate how the fact that the child is taught to write with the right hand has an important bearing on the lead which in the great majority of persons the left hemisphere takes over the right. As a general rule, the difference between the daily work of the two arms is considerably less than the corresponding difference in function between the right and the left speech areas, a difference in large part determined by the exercise of writing.

We believe, then, that there exists in all normal individuals a potentiality of development of speech centres in both halves of the brain, and that as the child grows and becomes definitely right-handed, there is less and less use of the right hemisphere areas for the purpose of speech, sensory or motor. It is questionable whether this potentiality of development of these right hemisphere regions ever entirely disappears. Long before the child has learned to write, and at a time when the majority of children may certainly be described as more or less ambidextrous, he is receiving auditory and visual impressions which there is little reason to suppose he is storing on one side more than the other. He hears thousands of words before he can himself speak ten, yet it is quite certain they are leaving their mark in his speech centres. The famous "Frost King" episode in the history of Helen Keller shows how the brain can store up what it does not understand, preserve the stored-up images, and utilize them years later as though they were original or intuitional, instead of having been in reality unconsciously absorbed.

Our hope of improvement in certain cases of aphasia depends on the belief that the right cerebral speech centres can be induced to take up functions hitherto incompletely exercised, if not entirely neglected; at the same time it must be clearly understood that the degree of possible utilization of these centres cannot be accurately foretold, and that therefore it would be unwise in all cases to assume the likelihood of their resuscitation. Some individuals are more right-handed than others; to some people the left limbs remain peculiarly "*gauche*,"

so that on the personal factor in these cases much may depend. Speaking generally, however, no case of aphasia should be abandoned as therapeutically hopeless without at least a serious attempt to awaken dormant activities in the opposite hemisphere. If these facts were only better known, the cultivation of ambidexterity would receive that impetus which it most assuredly deserves.¹

Differences in the Speech Mechanism in Different Individuals.—It is desirable to bear in mind that the description already given of the localization of the four main speech centres in the left cerebral cortex, in right-handed people, is intended to apply to individuals of ordinary education and development. Certain qualifications, however, must be made for exceptional cases.

Thus in the case of illiterates it is evident that the description cannot apply in its entirety. Weber quotes the instance of two right-handed illiterate Russians, who after a lesion in the right hemisphere (involving of course the left half of the body), entirely lost their ability to read, though they could still write. As they could read and write only to a limited extent, normally, the presumption is strong that the speech centres were bilaterally represented, though not much developed, so that a lesion involving the right side of the cerebrum, in the appropriate area, destroyed the function of reading, such as it was, the left side being too feebly developed to undertake the work. That their power of writing was not affected is explained by its unique representation in the left hemisphere. And there are cases on record where asphasia has not occurred, though the situation and size of the lesion might have led one to suppose its occurrence probable, presumably owing to the illiteracy of the subject and the lack of full development of the specialized centres.

Again, it has been pointed out in preceding paragraphs how extremely important is the accurate determination of the right- or left-handedness of the patient. Apparently anomalous cases of so-called "crossed aphasia" have been put on record, where though the patient was right-handed a left hemiplegia was accompanied with aphasia; according to the recent researches of Stier the patients in these cases have all

¹ In an article on the prophylaxis of aphasia (*Practitioner*, August, 1909, p. 238) Coley has the following remarks:

"I would suggest that provision should be made against aphasia in cases where, from the presence of granular kidney or other indications, there is reason to expect hemiplegia. My suggestion is that a graphic centre should be made to develop in the right side of the brain by practising writing with the left hand. The required development of the right graphic centre is not acquired until by practice the movements of the left hand in writing have become instinctive, as those of the right hand are. When this is the case, there is good reason to believe that no lesion of the left side of the brain would deprive the patient of the power to write. And it is far from impossible that power of speech would also be retained, because of the close functional connection between the speech centre and the graphic centre. Education of a graphic centre in the right side of the brain is a very different matter from the same thing as a method of treatment commenced after hemiplegia has occurred."

For reasons which are given below, however, it may be questioned whether such a right graphic centre would be autonomous, or functionally independent of the corresponding centre in the left hemisphere.

been potentially left-handed, but have been educated to use the right hand. There is no case reported of a frankly left-handed person having had his speech centres in the left hemisphere.¹

Another matter in regard to which there are individual differences concerns the recall of word-images and the method by which the executive speech centres are awakened to activity. There can be no doubt that in the child the auditory speech centre is the first to be called into action, and that it develops in advance of the other speech centres. It has already been stated that in the young child both auditory centres probably develop simultaneously, and Bastian believes that in the great majority of normal people recall of images in the auditory centres precedes and is essential to, the activity of the motor speech centre. When, therefore, a lesion cuts off the tracts from the auditory centre on the left side to Broca's centre, or when these two centres are in any way separated, it is possible that the right auditory centre takes up the burden and the importance of this from the point of view of prognosis will not escape the reader. Such persons as employ their auditory centres for the recall of words are known as "auditives," while in others the visual centres may be so strongly developed that the recall of word-images takes place in these rather than in the former; such individuals are said to be "visuals." In the consideration of a given case it is always desirable at least to attempt to discover whether the patient is in the habit of depending on his auditory or on his visual centre for the recall of words. Moreover; from the point of view of recovery from agraphia, much depends on whether the patient is a "visual" or an "auditive."

The Matter of Diaschisis.—It not infrequently happens that there is considerable difficulty in furnishing a satisfactory explanation of the relation of the clinical symptoms of aphasia, in a given case, to the site of the lesion as subsequently determined. Sometimes the symptoms disappear while the lesion remains; sometimes they persist, although the lesion is found not to involve speech centres directly; there is often considerable variability in the symptoms produced by similar lesions in different cases, and, finally there is sometimes striking dissimilarity in the degree of mental impairment presented by cases where the lesions are more or less identical. In order to explain these and other anomalies, as they seem, von Monakow has enunciated certain principles concerned with what may be called "action-at-a-distance." To this special form of action he gives the name "diaschisis," and defines it as "the temporary suspension of function which arises from the local interruption or disturbance of a tract of fibers which directs or carries out the function of a neighboring part." Secondary centres, not actually involved in the lesion, may not be stimulated from the destroyed centre, hence ensue certain impairments of function attributable to defects in regions which may be at some little distance from the actual disease.

¹ Mendel (Neurolog. Centralbl., February 1, 1912) has reported a case, with autopsy, of what is apparently right-brainedness in a right-handed individual.

It is important to note that von Monakow especially distinguishes these results of "diaschisis" from those due to accompanying phenomena of a circulatory, mechanical, toxic, or other type. As Bruce says: "If in spite of great extent of original lesion there is more or less complete recovery of speech, this depends not on vicarious intervention or education of corresponding convolutions of the other side, or any other part of the cortex, but from a retrogression of the original diaschisis. The conception shows that for the mechanism of speech much more extensive cortical areas are involved than has hitherto been assumed, but that the speech function can be easily affected by injury to certain sharply localized groups of bundles and cortical areas by diaschisis." Whether the conception, thus briefly sketched, can be called on to explain all anomalous symptoms in aphasic cases, is nevertheless debatable, but it is desirable that the reader should appreciate the possibility of diaschisis having been at work where an originally extensive aphasic defect clears up, to leave a small persisting residuum.

PROGNOSIS IN APHASIA

It is a difficult matter to say in any given case of aphasia what the outlook really is. Some originally severe cases clear up to an unexpected degree, others, slight from the beginning, remain uninfluenced. The complexity of the implicating factors is frequently considerable, yet none of them can be ignored in estimating prognosis. Certain considerations, however, will guide the physician in this connection.

Age of Patient.—Much will depend on the age of the patient. It is generally supposed that defects of speech occurring in early life are of less import, from the prognostic point of view, than if they occurred late in life, and this is, no doubt, true in the main. In children or in young adults whose speech centres have developed and been in functional activity before the onset of the disease producing aphasia it may be said that the outlook is good. I have seen aphasia well recovered from in the case of a girl, aged seventeen years, with right hemiplegia secondary to rheumatic endocarditis. On the other hand, a case has come under my notice of a child, aged five years, who has as yet made no detectable commencement of recovery from a right hemiplegia and aphasia of encephalitic origin, some fourteen months after the illness; up to its onset he prattled freely like any other child. Bateman quotes a not dissimilar case recorded by Duval, where a little boy of five made absolutely no recovery from an aphasia produced by a traumatic subdural hematoma of the left frontal region, a period of thirteen months supervening between the injury and his accidental death.

When the aphasia is a true congenital aphasia, *i. e.*, when there is no history of the youthful patient having spoken at any time, it is peculiarly arduous to state a prognosis. Apart from cases of idiocy and imbecility, which are not in question in this article, a child may show no signs of making any headway in learning to speak, read, or write, although

otherwise he would be passed as normal. Sometimes patient and long-continued treatment will effect an improvement, will even apparently develop the faculty when that has been entirely wanting. Wyllie, for instance, quotes the case of a boy, aged seven years, whose hearing was almost normal but who had no power of speech. His general intelligence was below the average. Exactly the same means were adopted as with a totally deaf child, and after four years' instruction he was able to speak very distinctly, but with slight hesitation. Sometimes, again, spontaneous recovery, or rather spontaneous awakening of speech, occurs in children who have never spoken at all, even up to the age of six years. Thus Bateman quotes the following case from Wigan: "A London merchant had a son, aged eight years, perfectly dumb, and the family had abandoned all hope of his ever being endowed with the gift of speech; there was no defect in the intellect nor lesion of any other faculty. In a water party on the Thames the father fell overboard, when the dumb boy called out aloud, 'Oh, save him! save him!' and from that moment he spoke with almost as much ease as his brothers." Bastian, too, narrates an equally striking case which came under his own observation. A child, aged five years, who had suffered from fits in infancy had never spoken a single word until one day, on the occasion of an accident happening to one of his favorite toys, he suddenly exclaimed, "What a pity!" though he had never previously uttered an articulate sound. The same words could not be repeated, nor were others spoken, notwithstanding all entreaties, for a period of two weeks. After that time, however, rapid improvement took place and the child soon became quite loquacious.

No doubt instances such as these are somewhat dramatic and will always perhaps remain infrequent. When it is remembered however, how patience and educational skill combined to triumph over almost insuperable difficulties in the way of the acquisition of speech, as in the cases of Laura Bridgman, Helen Keller, and others, one hesitates to condemn any child, however backward in talking (provided he is not demented or idiotic) to life-long speechlessness. Due reference to methods will be made subsequently.

When we come to adults, on the other hand, the prognosis is, of course not so good. Adult acquired aphasia may never be recovered from. Every practising neurologist must have had experience of cases of motor aphasia in which no improvement ever occurs. We shall see, however, in subsequent paragraphs how desirable it is not to rest resignedly contented with such a state of affairs when cases have been put on record of considerable amelioration under suitable treatment.

Again, it may happen that even although the patient is well up in years spontaneous improvement may set in. Bastian, for instance, quotes a case published as long ago as 1865 by Banks, where a gentleman, aged fifty-four years, after six years of motor aphasia, gradually improved, acquiring new words and finally being able to converse on ordinary subjects. As a general rule, the longer the interval between the ictus and the reawakening of speech the less likely is speech to be

abundant. It is in the cases where the patient is able to articulate some words again within, say, some weeks of the onset that the physician may venture to give an encouraging prognosis.

Severity of the Attack.—Much, further, depends on the severity of the attack of aphasia. In this connection it is noteworthy that there is a large class of cases where the aphasia may fairly be described as transient, and in these, naturally, the prognosis is good. Temporary disability in speaking—and it should be clearly understood that although reference is here made chiefly to defects on the motor side the conclusions hold good for sensory aphasia also—is met with under varying conditions. It occurs after certain attacks of Jacksonian epilepsy, in migraine, in what seems to be spasm of cerebral vessels, in incomplete thrombosis or in endarteritis obliterans, in arteriosclerosis, in certain toxic conditions, in various toxi-infective diseases, and of course in hysteria and insanity, to which reference is not here made. The physician may find it difficult to realize how easily cerebral speech mechanisms may be disturbed by transient influences. A case that came under my own notice when I was Resident Medical Officer to the National Hospital, Queen Square, London, impressed me considerably at the time. A woman with a left Rolandic tumor was successfully operated on, and made a good recovery as far as motor speech was concerned, being able to talk with comparative readiness some three weeks after the operation. As there was very slight leakage from a corner of the scalp wound I put in two single stitches, closing the minute orifice. For forty-eight hours thereafter the patient was completely aphasic, presumably because of alteration in the tension of cerebrospinal fluid as a result of the closure of the wound. If so trifling a circumstance could interfere with the function of the cortex to the extent it did, it can well be understood how transient aphasia may result from passing ischemia, claudication, arterial spasm, and so on, and it is indeed conceivable that the aphasia sometimes associated with profound emotion may rest on a basis a little more material than that of mere psychical disturbance.

In all such cases, the prognosis is satisfactory enough. Be the duration of the aphasia what it may, within limits, one may still be confident that recovery will ensue. Of course, it behooves the physician to make an exhaustive examination of the patient, for not otherwise can an accurate diagnosis be reached. It would be unfortunate if imperfect examination should lead to a confident prognosis in transient aphasia occurring, say, as one of the symptoms of the congestive attacks of general paralysis. Focal symptoms in that disease are far from uncommon, and occasionally temporary aphasia may be noted when the more usual somatic signs of general paralysis are still incompletely developed. It should not be necessary, either, to warn the reader of the possible contingency of transient aphasia from cerebral vascular disease establishing itself definitely when an ictus occurs. As a rule, however, it should not be difficult to reach a diagnosis with that degree of accuracy which will obviate mistakes of this kind.

When an aphasia is definitely established in a patient who is of middle age or beyond, severe in degree, and apparently stationary, prognosis should always be guarded. Certain considerations will guide us in this connection, although they are of the most general description and must not be held to be absolute in their value.

(a) If there is little or no indication of generalized cardiac, arterial, or renal disease the outlook is better than where there are degenerative changes in these systems.

(b) If the aphasia is uncomplicated by any paralytic symptom the chances of functional restoration are better than if there is marked hemiplegia. While this is a fairly good general rule there are, needless to say, discouraging exceptions. The most remarkable case of sensory aphasia that has ever come under my notice was unaccompanied by any paralytic phenomena, but no recovery was effected. Palsies are simply an index of the widespread nature of the lesion, and, other things being equal, the larger the lesion the less likely is marked improvement.

(c) Bateman holds that the prognosis is more favorable when loss of speech depends upon direct injury to the brain, but I have not been able to convince myself of the truth of this. Thus in a case where hemiplegia and aphasia resulted from an iron lid falling on the patient's head from some height, operation revealed a subdural hematoma of considerable extent; its removal, about two years after the accident, was not followed by any material improvement. There is a type of case in which "cortical degeneration" (as one calls it, *faute de mieux*) ensues on traumatic lesions of the cerebrum, sometimes even, as I have seen, from operative trauma, and under these circumstances the hope of amelioration is reduced to a minimum.

(d) It is unwise to deduce anything from a consideration of the nature of the onset of the aphasia, *i. e.*, whether sudden or slow. Older writers, such as Winslow, held that it was unusual for sudden speechlessness to occur without being immediately followed by acute cerebral symptoms. But there is no inherent reason why a sudden aphasia should not be recovered from as readily as a slowly oncoming aphasia; in fact, the latter type is more commonly indicative of a graver intracranial condition than the former, *viz.*, cerebral tumor. A progressive hemiplegia and aphasia is of more serious import on this account.

Bastian has drawn special attention to two sets of considerations which have a direct bearing on the prospects of recovery from speech defects. They are described respectively as "functional restitution" and "functional compensation." By the former is meant the possibility of the actual lesion undergoing diminution, so that parts of the brain which have been damaged may resume functional activity. More than one indirect allusion has already been made to this matter. The establishment of collateral circulation on the confines of a thrombosed area may lead to the disappearance of some at least of the symptoms. Absorption of products of inflammation or disintegration may be followed by a degree of functional restitution. Removal of pressure may also have a beneficial result. In a word, where there is inter-

ference with, rather than destruction of, neuronie activity, restoration of function is always possible if the obstructing agent be removed.

By functional compensation is meant the development of auxiliary speech mechanisms in undamaged parts of the hemispheres, usually, presumably, in the corresponding areas of the right hemisphere. Reference has already been made to this question of compensation. There can be no doubt of the reality of this method of recovery in certain instances, but, as Bastian truly observes, "the difficulties in arriving at a conclusion as to the relative degrees of curability of the different forms of speech defect by a process of functional compensation are extreme," due in part to the impossibility of estimating the value of the personal factor in any given case, in part to uncertainty as to the plasticity or otherwise of the individual's nervous tissues, and so on. Anyone who is familiar with the literature of the result obtained in the treatment of aphasia will have had this fact impressed on him, that some cases do unexpectedly well, while others make no advance in spite of every stimulus and allurements.

THE TREATMENT OF APHASIA

In every case of aphasia, treatment must be prosecuted along two lines, viz., the treatment of the morbid process underlying the phenomena, and the treatment of the symptomatic condition once it is established and more or less stationary. These lines of therapeutic endeavor must be kept apart, in the physician's mind, and handled separately in an article such as the present.

Treatment Directed to the Underlying Morbid Process.—The commonest causes of aphasia are embolism, hemorrhage, thrombosis, inflammations, and tumors, and these may be considered in turn.

Embolism.—It is unnecessary to dilate on the diagnosis of cerebral embolism in this place, and the reader is referred elsewhere for a full discussion of its treatment. The following remarks, therefore, will be very brief and general in character. The patient should be kept absolutely at rest, and if he is unconscious the condition of the bladder and rectum must always receive attention. Careful feeding, with non-stimulating liquid food, must be persevered with till the return of consciousness. Purgation is always undesirable, for cerebral overdepletion may aggravate a tendency to thrombosis around the embolic focus. As a rule, stimulants are uncalled for, but in this matter each case must be treated on its own merits. The after-treatment of the patient is as important as the early treatment.

Hemorrhage.—The proviso mentioned above applies to the question of hemorrhage also, and to the topics of the subsequent paragraphs. The main indications where the diagnosis of cerebral or intracranial hemorrhage has been made are (1) to stop the hemorrhage, and (2) to reduce or relieve the increase in intracranial pressure. The patient must be kept absolutely at rest, the head and shoulders slightly raised,

and the head turned to one side. Certain drugs have been vaunted as tending to increase the coagulability of the blood, in particular the calcium salts, but their efficacy is doubtful. Arterial pressure can be lowered by venesection, or by purgation, and for a consideration of the subject the reader is referred to another article. Once the acute stage is over, and the risk of further hemorrhage diminished, the patient will still require unremitting attention for some time. Rest and tranquility are always indicated; excitement and exertion must be studiously avoided. Non-stimulating diet must be ordered, erring always on the side of administering too little. The blood pressure may be used as a gauge to the patient's condition. No reference is made here to the important question of surgical interference in intracranial hemorrhage.

Thrombosis.—The physician will direct his efforts toward lessening the coagulability of the blood and preventing the extension of the thrombotic process. For this purpose the alkaline citrates or citric acid may be exhibited with advantage. In addition, a certain amount of circulatory stimulation is desirable, its object being to increase blood flow in smaller vessels, thus limiting, as far as may be, the thrombotic process and its results, and at the same time to minimize the possibility of further thrombosis. For this purpose strychnine, digitalis, etc., may be recommended. Anything which tends to lower the blood pressure or lessen the heart's force must be avoided. Hence active purgation is uncalled for, if not absolutely contraindicated. It is established that iodide of potassium is worse than useless except in frankly syphilitic cases; and in the latter even its use is often barren of result. It should never be given except with, or after, a thorough course of mercury, and it should be coupled with suitable cardiac stimulants.

Once the immediate danger of thrombotic extension is over, the patient's life must be assiduously ordered by the physician, according as the morbid agent is syphilis, arteriosclerosis, and so on.

Inflammations.—Encephalitic or meningo-encephalitic processes, acute or subacute in type, are treated on the lines of infective conditions in general. Where the disease is febrile in character, rest in bed, continued for a week or more after the cessation of the fever, is essential. Any simple aperient should be used regularly. A liquid diet is desirable, and small doses of the tincture of aconite, or of salicin or salol, may be administered. Urotropin may be given empirically. It is questionable whether counterirritation over the nape of the neck or behind the ears is of any efficacy. Sometimes repeated lumbar puncture may be of value. Once the acute stage is over treatment must be pursued on general lines, and as the patient is often rather prostrated, due regard must be had to the matter of stimulation.

Tumors.—The medical treatment of intracranial tumors is practically valueless. Even in frankly syphilitic or gummatous cases treatment with antisiphilitic remedies is not infrequently unaccompanied by any alleviation. One must therefore look to the surgeon for assistance,

but any further discussion of this topic is out of place in the present article.

Reference has already been made to the occurrence of transient attacks of aphasia, more particularly in the subjects of cerebral vascular disease, attacks which are usually indicative of temporary meiotopia and may be regarded as a danger signal of the possibility of such lesions as have just been described. Patients who have already developed a cerebral hemiplegia frequently suffer from epileptic attacks in which aphasia from exhaustion of the corresponding centres may be one of the symptoms. In the former case treatment must be pursued by the adoption of measures that have been already sketched. In the latter case, suitable administration of sedatives will usually suffice. Sodium bromide, given at night, is often all that is required. Similarly, an aphasia may be recovered from, but reappear as an exhaustion phenomenon after cerebral discharges, and the same treatment is here applicable. Occasionally, also, it may be difficult in a given case to distinguish between a transient aphasia from an epileptiform discharge, and a fresh attack from a recurrent thrombosis; but if treatment be directed both to the epilepsy and to the thrombosis, it will be greatly to the patient's benefit.

Treatment Directed to the Restoration of Speech by Functional Compensation.—This is one of the most important parts of our subject, and it is also one on which opinion is to a great measure divided. A number of cases have been put on record where the organs of speech were reëducated *de novo*, with surprisingly good results, but not a few writers on the subject remain unconvinced. Niessl von Mayendorf, for instance, says:¹ "Wenn nach einem Vierteljahr noch keine sprachlichen Rudimente produziert werden, muss man mit Wahrscheinlichkeit unheilbare Stummheit befürchten." And again:² "Diese Behauptung allerdings steht im Widerspruch mit den Erfahrungen Gutzmanns, welcher selbst nach jahrelangem Bestehen der motorischen Aphasie durch seine Methode Besserungen erzielt hat. Ich will diese Erfolge durchaus nicht in Frage stellen, möchte nur betonen dass Fälle mit jahrelanger persistierender motorischer Aphasie ohne alle Rückbildungsvorgänge jedenfalls zu den schweren, prognostisch ungünstigen, und Besserungen, oder sogar Heilungen zu den grössten Seltenheiten gehören." From which it may with fairness be implied, I think, that Niessl von Mayendorf does not think these methods very promising; or their application worth while. André Thomas, on the other hand, speaks much more hopefully. Applying in a case of motor aphasia methods more or less analogous to those of Gutzmann with

¹ When after three months still no rudiments of speech can be produced, one must fear in all probability an irrecoverable aphasia.—Ed.

² This assertion certainly stands in contradiction to the experience of Gutzmann, who has obtained improvement by his method in motor aphasia of several years' duration. I will not bring these results in question, but would like only to note that cases of motor aphasia persisting for years without deterioration belong at all events to the severe cases of unfavorable prognosis, and improvement or recovery is very rare.—Ed.

a considerable degree of success, he remarks:¹ "Sous l'influence de la rééducation, la parole est revenue en grande partie, les mots sont correctement articulés, chaque syllabe est bien détachée, aussi bien dans la parole spontanée que dans la lecture à haute voix. En présence de pareils résultats obtenus dans un cas d'aphasie motrice corticale datant de cinq ans, il y a lieu de penser que le même traitement appliqué dès les premiers mois, chez les malades atteints d'aphasie motrice corticale, est suffisamment indiqué." And there are other observers whose experience is sufficiently encouraging to warrant the assertion that the physician of today ought not to relinquish hope until such methods have been at least essayed.

One or two instances of improvement or success by this means may briefly be quoted.

Bristowe records the case of a man, aged thirty-six years, who after a series of epileptic fits became entirely aphasic, and at the end of seven months he was still unable to utter a single articulate sound. He understood all that was said to him, and had no word-blindness; he was also able to conduct a conversation by writing. As he was able to perform all ordinary movements of the lips, tongue, jaws, etc., and was also capable of vocal intonation, Bristowe concluded that this inability to speak was probably due to his having forgotten how to combine automatically the movements of these organs. "He informed the patient of his views of his case, which the latter appeared to understand perfectly. He explained to him that ordinary vocal sounds are composed of two factors, namely, laryngeal intonation, which he was still able to produce; and articulation effected by the lips, tongue, and associated parts, of which he was as yet incapable. He then got him to sound a laryngeal note, subsequently explaining to him and showing him how to modify the shape and size of the oral passage and aperture; and getting him at the same time to expire, either with or without laryngeal intonation, he made him sound, both in a whisper and in a loud voice, certain of the more simple and obvious vowel sounds.

At his next lesson he set to work to teach him the labials, and he subsequently taught him by the same process the lingual and guttural consonantal sounds; and thus, in the course of four or five lessons, principally by making the patient watch the movements of his [Dr. Bristowe's] lips, he regained the power of articulating all the simple vowel and consonantal sounds. He then began to teach him to combine letters, and eventually succeeded in making him talk well, although he spoke somewhat slowly and evidently had to give more thought to the pronunciation of his words than healthy people ordinarily do."

Dr. Bristowe adds: "The lessons which I gave him were, as I have

¹ Under the influence of rééducation speech returns in large part, the words are correctly articulated, each syllable is well detached, as well in spontaneous speech as in reading. In the presence of parallel results obtained in a case of cortical motor aphasia of five years' duration there is reason to think that the same treatment applied in the first months in cases of cortical motor aphasia is sufficiently indicated.—ED.

shown, few and short. But he himself, as soon as ever he had appreciated the fact that he had organs capable of evolving articulate sounds, supplemented my instruction with the most zealous practice. Thus the vowels and consonantal sounds which he uttered somewhat imperfectly during a lesson were learnt accurately by my next visit; and as soon as he had begun to combine sounds, he practised them in various combinations with great industry; the sister of the ward and nurses, and more especially three or four intelligent patients who were friendly with him and interested in his progress, giving him constant assistance."

This case, no doubt, was singularly uncomplicated, and the aphasic defect was definitely limited, nevertheless there can be no doubt that reëducation as a therapeutic measure was thoroughly justified by the event.

In commenting on the case, Bastian, who also quotes it, remarks that the method requires some skill in the teacher, and not a little enthusiasm and perseverance, to carry it to a successful issue.

Reference has been made to Thomas' paper on this subject, and to the good results he obtained. One of his cases is the following:

A woman, aged thirty-four years, with right hemiplegia and aphasia consequent on exploration of the brain for a supposed cerebral abscess, entered the Salpêtrière some five years after the onset of the condition, and it was found on examination that she suffered from complete motor aphasia, the only words she could utter being "oui" and "non." She could not repeat words, and could not read aloud. She could write only her name and her age with her left hand, and was not able to write to dictation, but she could copy. Reëducation was commenced by teaching the patient, who observed the movements of tongue and lips, to repeat vowels and simple syllables (association of a consonant and a vowel). A fortnight later she could articulate all syllables, and then she relearned spelling and the reading of simple syllables by the same method; at the same time she made attempts at writing. Once the constituent elements of words were regained, she learned to repeat words of one syllable, then of two, and of several syllables; she learned to read words in a syllabic fashion, and to repeat syllables composed of two consonants and a vowel. Reëducation ceased at the end of six weeks, and the patient was left to herself. After a year, she was able to reply to any sort of question, using words correctly articulated and without hesitation; she could not compose sentences, but she could make herself understood. She could repeat short phrases quite well, but long sentences were repeated only in part. She read aloud correctly articulating each syllable distinctly. Her understanding of what she read was much improved, but the power of writing spontaneously and to dictation had not altered much; she had not, however, made many attempts in that direction.

A case published by Wyllie may also be briefly referred to. It was one of motor aphasia without agraphia, and the reëducation is described as follows: "We did not trouble the patient with the names of the letters, but taught him from the beginning the letter sounds of the

physiological alphabet. In doing so, we adopted what may be called the "mother's method." Beginning with the labials, we taught him to say papa, apap, appa, thus giving him the consonant p as an initial, a terminal, and a mid letter. Then we taught him to say baba, abab, abba; then mama, amam, amma, and so on throughout the alphabet. He was shown by lip reading how to place the lips, tongue, etc., for the pronunciation of each letter sound. Being an intelligent and diligent pupil, he soon was able to go over the whole of the letter sounds in this triple fashion, from one end of the alphabet to the other, by heart, without looking at the paper. We then supplied him with the children's books known as the *Little Primer*, and the *Little Reader*, and so on," etc.

In this case the ultimate result was very good.

Similar or analogous cases have been published by Hun, Schmidt, Bateman, Mills, Goldscheider, Gutzmann, etc.

It will be recognized, then, that the evidence favorable to the reëducation method is ample enough to render its comparative neglect by the profession rather inexplicable. Naturally, in order that it may be prosecuted to a successful issue, the combination of a suitable case, a diligent pupil, and an enthusiastic and patient teacher is almost a *sine qua non*, and doubtless such a combination is not always to be found. Yet even a modicum of success is worth striving for, and it may be hoped that henceforward the physician will address himself to the task with greater devotion and assiduity.

In all cases the method of reëducation is essentially the same, and it is based, as Collier says, upon the processes by which a child first learns the elements of his speech, the details being varied according to the nature of the speech defect in each patient.

Methods of Reëducation.—A sketch may now be given of the actual methods in vogue, and in this matter the writer is indebted in part to an excellent paper by C. K. Mills, on the treatment of aphasia by training. These pedagogic methods may be classed provisionally as:

1. The repetition method: The patient is taught to repeat words after others, and later spontaneous recall becomes possible as the patient improves. It is associated with allied methods such as reading aloud, copying, and writing from dictation.

2. Phonetic methods, such as the method of the physiological alphabet suggested by Wyllie, and the use of phonetic readers.

3. The lip-reading method: the patient learns to imitate the movements of articulation, enunciation, and vocalization either from observing the lips and tongue of others, or by observing himself in a mirror.

4. Various special methods, *e. g.*, that of Goldscheider, to be referred to later, in which the patient is trained to repeat meaningless syllables.

Repetition Method.—Mills' own application of the repetition method is after the following style: Repetition of letters, words, phrases, and sentences, recognized by the patient in reading or repeated by him after others; repetition of names of objects seen by him, or the naming spontaneously of such objects either seen or made known to him through

his other senses, *e. g.*, by touch; repetition of writing, either copying, or spontaneously, or from dictation.

Dana's repetition methods are here quoted *in extenso*:

(a) "Repeat five exclamatory words, such as 'ah,' 'oh,' or other exclamations expressing joy, anger, or other emotion. Repeat after teacher ten monosyllabic nouns or pronouns. Repeat ten polysyllabic nouns. Repeat ten verbs. Each time a noun is named, let the patient see the object, feel it, and see the written or printed name on a piece of paper before him, thus stimulating visual, auditory, and tactile memories at the same time.

(b) "Repeat the letters of the alphabet, these letters being held in front of him. Repeat letters of the alphabet after writing and looking at each one. Repeat the numerals from one to twenty. Repeat them while looking at the figures. Write and repeat the figures.

(c) "Repeat ten simple qualifying adjectives, such as white, black, red, smooth, soft, rough. At the same time, let him see the object and color, or feel the same.

(d) "Later, let him try to repeat sentences of three words in which the noun is joined to the adjective, *e. g.*, 'book is red,' 'box is white,' etc.

(e) "If the patient has any musical capacity, sit him at a piano and hum the notes of the piano, going through an octave, and then let him try to hum a tune, striking a note at the same time. Finally teach him to sing the tune through, and then introduce the possible words. Some patients can sing before they can talk.

(f) "Copy sentences made up of the words he is being taught. Let him have an ordinary copybook and have the copy at the top of the page. Let him fill a page every day, trying at the same time to pronounce the words as he writes them. Have him copy first the familiar nouns, and then the simpler verbs, then the simple adjectives; finally, let him copy sentences. Take a small vocabulary and repeat from this, not trying to enlarge too soon.

(g) "Write the letters of the alphabet and as the patient writes them get him to try to repeat them. Do this without a copy, if possible. Then let him write words to dictation, using the same vocabulary above referred to. Finally, let him try to write short sentences to dictation, then try to read them after he has written them, with assistance at first, then without.

(h) "Write numbers one to twenty and say them out loud when written.

(i) "The patient should allow himself to be read to, for a short time, twice a day, and he should also try to read a quarter of a page every day."

Phonetic Methods.—WYLLIE'S METHOD.—Of phonetic methods, properly so-called, Wyllie's is probably the best known. Apropos of the case cited above (page 491) Wyllie says: "In training a patient with motor aphasia to speak, it is often of advantage to him to pronounce simple words in his hearing and ask him to repeat them. It is largely

by practice in such repetition of words heard that the patient, in so many cases, reacquires the power of speech. But I think the process may be expedited if it is conducted on scientific principles. It should be remembered that the patient has lost the power of uttering even the simple letter-sounds. If he can be got to master the letter-sounds in the first instance, it will be more possible for him, afterward, to produce their combinations in the form of words." For this purpose Wyllie has invented a physiological alphabet, which is here reproduced. In using it the patient is taught the letter-sounds of each vowel and consonant (into nearly all the consonants a vocal element enters). An illustration of its use will be found in Wyllie's own case of motor aphasia, already quoted.

WYLLIE'S PHYSIOLOGICAL ALPHABET

Illustrative Sentences

I. VOWELS

v—i e a o u—w

These should be pronounced in the Latin manner, as *ēē*, *eh*, *ah*, *oh*, *oo*; *y* and *w* are consonants, not vowels, but they have very close relationships to the vowels, initial *y* being very closely related to *i*, and initial *w* to *u*.

I. VOWELS

Even ancient elves are awed over ooking.

This sentence represents only long vowels. Their short equivalents can be represented by attaching the letter *t* to each vowel, thus:

ēēt, *it*, *et*, *at*, *ut*, *ot*, *ōōt*.

II. CONSONANTS

II. CONSONANTS

	Voiceless oral consonants.	Voiced oral consonants.	Voiced nasal resonants.	
Labials	<i>P</i>	<i>B</i>	<i>M</i>	Peter Brown made white wax.
(First stop position) .	(<i>W</i>)	<i>W</i>		
Labiodentals	<i>F</i>	<i>V</i>	<i>..</i>	Fine villages.
Linguodentals	<i>Th</i> ¹ <i>S</i>	<i>Th</i> ² <i>Z</i>	<i>..</i>	Thinkest thou so, zealot?
Anterior linguopalatals	<i>Sh</i> <i>T</i> (<i>L</i>)	<i>Zh</i> <i>D</i> <i>L</i>	<i>N</i> <i>..</i>	She leisurely took down nine large roses.
(Second stop position) .	<i>..</i>	<i>R</i>		
Posterior linguopalatals	<i>K</i> <i>H</i> or <i>Ch</i>	<i>G</i> <i>Y</i>	<i>Ng</i>	
(Third stop position) .	<i>..</i>	(<i>R</i>)		Can Gilbert bring Loch Hourn youths?

The voiceless *W* and the voiceless *L* have been given above within brackets, the former being now almost confined to Scotland, and the latter being peculiar to Wales. The burring or uvular *R* is also given within brackets.

Lip Reading.—The reader will have observed that Wyllie's phonetic method is really combined with the third method, to which allusion has been made, viz., that of lip-reading. The patient is shown by direct lip-reading how to place the lips, tongue, etc., for the pronunciation of each letter-sound. Wyllie points out that lip-reading has not hitherto been perhaps so successful in England as it has been in Germany, chiefly because the English language is not so well fitted for this as the German. This depends on the fact that in English the spelling of the words have departed so widely from their sounds; spelling is much less strictly phonetic in English than in the German language. Nevertheless, the system of lip-reading is coming more and more into vogue, and as an adjunct to the reëducation of aphasics it is invaluable. The reader is referred to special text-books and articles on the subject for further information.

GOLDSCHIEDER'S METHODS.—Goldscheider's methods are as follows: The patient commences with the simplest vowels and the explosives, and articulates Pa, Pe, Po, then Ba, Be, Bo, and so on. Then the consonants N, F, W, etc., are utilized in the same way. If he is unable to repeat sounds as they are, he must employ lip-reading, and endeavor to imitate sounds in this way.

After learning to associate vocal sounds with speech movements, he next proceeds to associate them with writing and reading, instruction being carried out methodically by letters, syllables, words, and sentences.

Words are next associated with their meaning; an object is held in front of the patient, and he is told what it is; he repeats the word, spells it, and picks it out by the use of an alphabet.

Goldscheider thinks that the best way to instruct in the tones and accents of words is by the patient's memorizing rows of meaningless syllables, the order of these being changed from time to time. He gives the following example:

ut	re	mi	
ut	mi	re	
re	ut	mi	
re	re	ut	
mi	mi	re	
mi	ut	ut	etc.

GUTZMANN'S METHODS.—Gutzmann's suggestions resemble those of Mills and Wyllie. Mills gives the subjoined *resume* of his methods:

1. "Repetition of letters, words, phrases.
2. "Repetition of sounds in particular letters, syllable associations, combining vowels with the labials, dentals, and other consonants.
3. "Tongue exercises are practised and tones are taught.
4. "The patient is made to imitate movements of articulation and enunciation by watching the facial movements of others, and to improve himself in the same respects by observing in a looking-glass his own

movements and expression in articulating. The association of vocalization with reading and writing as well as with speech is practised; in other words, the patient learns to read aloud, and also to repeat what he writes; geometric forms are taught, and the aphasic writes from dictation, from copy, and spontaneously. Words are associated not only with their meanings but a suggestion is made to repeat meaningless syllables in various combinations. Names and concrete objects are associated."

It will be noticed, from a consideration of these varying methods of reëducating patients afflicted with aphasia, that in no one of them is attention confined solely to repetition, or to lip-reading, or to phonetic instruction. It is a conviction based on experience that a judicious combination of different plans of attack is likely to be followed by the most satisfactory results. Reference has been made above to the case of Helen Keller. If the reader will turn to the appendix in the *Story of Her Life*, written by herself, he will there find what is to the physician the most interesting part of the book, viz., the series of letters written by her teacher, in which the latter describes her pedagogic endeavors, and enters into the details of her teaching. Inasmuch as the pupil was both blind and deaf, to render her finally capable of speech was indeed a triumph, yet it was but by the application of methods not essentially dissimilar from those here given that that wonderful result was attained. If the effort to reëducate aphasics to speak were attempted more frequently and more patiently, there can be little doubt that the record of successes would ere long be notably enriched.

Hitherto, in the matter of functional compensation, we have directed our attention more particularly to cases of motor as opposed to sensory aphasia. Gutzmann seems to be of the opinion that sensory aphasia is more difficult to treat than motor aphasia, from this standpoint. It may be so. In a case of auditory aphasia, for instance, it is clear that treatment must be given such as is calculated to develop the auditory word centre in the right hemisphere, and to bring it into more complete relation with Broca's centre in the left. Bastian recognizes that the process must entail the expenditure of much time and patience, but by concentrating the patient's attention on simple vowel sounds, and afterward on monosyllables, spelled letter by letter, a start may be made, and once this is the case we may expect improvement to become more rapid. If the patient has not any accompanying visual aphasia, reading will no doubt aid in the determination of direct functional compensation by new cerebral paths. A good example of amelioration along these lines, in a case of word-deafness, was recorded by Schmidt and is quoted by Wyllie.

Word-blindness.—Similarly, in word-blindness, the patient must be treated as a child learning to read for the first time, especially if he is letter-blind as well as word-blind. I cannot do better than quote Bastian's suggestions for the treatment of this type of sensory aphasia.

"The patient's attention must be concentrated upon individual letters one by one, at the same time that the teacher pronounces their

names. After a certain amount of practice of this kind the patient must be encouraged to try and name the letters himself (making use here of the oral method), in order to endeavor gradually to build up a commissural and functional relationship between the right visual word centre and Broca's region—as in the deaf-mute who is taught to speak. Then from letters the teacher should pass to monosyllables, naming each individual letter, and following it by the pronunciation of the sound as a whole. Here again efforts to read such monosyllables should be made by the patient (the teacher assisting by the oral method) until some facility has been acquired. Then an advance must be made with words of one and two syllables. Exercises of this kind carried out methodically and over a long period will be necessary for this education of the right visual word centre, and for the establishment of functional relations between it and the two undamaged word centres in the left hemisphere, viz., the auditory word centre and Broca's centre. At this period, or earlier, efforts should also be made to call the right visual word centre into further activity, by teaching the patient to write with the left hand."

To show the reader that treatment of this sort may be carried out with the greatest success, I may refer him to the latest work on the subject—a paper read by Dr. Hinshelwood, of Glasgow, at the Annual Congress of the British Medical Association at Liverpool, July, 1912. One of his patients was a woman, aged thirty-four years, who had been completely word- and letter-blind for fourteen months; she also had right homonymous hemianopia. A school-master took great interest in her reëducation. It was found that the effort of education was great; ten minutes at a time proved to be enough. Ultimately the patient learned to read simple Bible texts by spelling out the words. After an interval of ten years, progress had been really excellent. She could read a newspaper fairly fluently, only occasionally being compelled to spell words. Another case was that of a girl, aged fourteen years, with right hemiplegia and aphasia of eighteen months' duration. When first seen she was completely letter-blind and had right homonymous hemianopia. Her auditory memory was unimpaired. Reëducation was started, and after learning the alphabet she was allowed to spell out words letter by letter. In four months she had made considerable progress and could recognize any letter and many small words. Longer words she had to spell so as to get the aid of her ear. Two years later she could read as well as ever.

Hinshelwood advocates strongly personal teaching in all cases, and says he has found a number of short reading lessons during the day to be better than one long one.

It was long ago pointed out by Hughlings Jackson, as remarked above, that a patient suffering from motor aphasia may be able to say "yes" or "no," but be quite unable to "propositionize" with them, *i. e.*, to use them correctly as the expression of a statement. Herein lies an important truth which concerns the subject of the reëducation of aphasics very closely. The faculty of propositionizing in language

is really the most fundamental defect of the aphasic; he has, as Mills remarks, the greatest difficulty in regaining just those parts of speech which are concerned with qualifying and correlating. Exactly the same difficulty was experienced in the case of Helen Keller. An infant making its earliest essays in speech will say "baby hungry" before it will say "baby is hungry," and long before it can say "I am hungry." The aphasic will manage "paper-white" long before he gets to "the paper is white." Prepositions and articles are under an eclipse, as it seems, or actually lost, and nothing shows the poverty of his language so much as the omission of these connecting, modifying, qualifying links. From the outset, therefore, the teacher must set himself to tackle these omissions seriously. In every possible way the manner of employing such parts of speech as prepositions, conjunctions, and auxiliary verbs should be impressed on the patient. When he indulges in incomplete sentences the complete sentence should always be repeated and explained. In fact, the grammar has to be relearned; it has to be studied after the routine method of the school. Otherwise, the ultimate result will still be very imperfect.

DYSARTHRIA AND ANARTHRIA

Dysarthria or anarthria may be caused by lesions either of the upper or of the lower motor neurone, and by unilateral, but more frequently bilateral lesions.

To begin with, there are certain congenital anomalies such as cleft palate or hare-lip which are associated with dysarthria, while other defects, such as imperfect teeth or local conditions in the mouth, fauces, etc., such as tumors, will of course impede correct muscular action and thereby produce dysarthria. Apart, however, from these, over which we need not delay, diseases of the lower motor neurone are often accompanied by dysarthria; disease of the extreme motor periphery, for instance, such as facial myopathy, and especially that type known as the Landouzy-Dejerine variety, has dysarthria as one of the symptoms. Lesions involving the seventh, ninth, tenth or twelfth cranial nerves, notably bilateral lesions, will produce defective articulation, and lesions of the corresponding nuclei in the medulla, as well as infranuclear lesions, will have the same effect. Included in this category are the familiar bulbar palsy, the bulbar type of progressive muscular atrophy or amyotrophic lateral sclerosis; also acute bulbar conditions, poliomyelitis inferior, and myasthenia gravis. In the former of these, acute embarrassment of medullary nuclei may supervene on a toxi-infective condition, and dysarthria or anarthria result. In the latter condition the dysarthria is essentially variable, depending on fatigue, and recovering with rest. Certain vascular lesions in this medullary area are deserving of mention; prominent among these is occlusion of the posterior inferior cerebellar artery on either side, minute branches from which enter the lateral aspect of the medulla and supply the intramedullary nerve trunks and nuclei of the ninth and tenth nerves.

Again, dysarthria may ensue when, with intact lower motor neurone, certain of the influences of other intracranial centres acting on it—the “final common path” of Sherrington—are impaired. Thus dysarthria frequently occurs in cerebellar disease; it is, as a matter of fact, better described by ataxia of articulation. Similarly in Friedreich's disease, disseminated sclerosis, and pontocerebellar, pontine, and pontomedullary tumors, the articulatory speech mechanism is liable to be disturbed from failure of coördinating controls. The dysarthria of paralysis agitans is another instance of a similar defect of extra-pyramidal origin; with intact lower motor neurone the patient is nevertheless dysarthric, owing to rigidity of the speech mechanisms without paralysis. This is especially well illustrated in the interesting familial nervous disease known as progressive lenticular degeneration. The dysarthria of general paralysis of the insane is in part of ataxic origin, in part due to certain central defects.

Coming to disease of the upper motor neurone, any interference in the path of the pyramidal fibers from the operculum, and lowest part of the precentral gyrus, by the genu of the internal capsule and the inner fifth of the crus, to the pontomedullary nuclei of the motor cranial nerves concerned in articulation, will produce disturbances of that function and result in dysarthria or anarthria. The morbid phenomena are much more apt to arise if the lesion is bilateral, and we then have the well-known “pseudobulbar paralysis”—an unfortunate name, for it is a true paralysis of bulbar muscles, just as much as the paralysis of the limbs in hemiplegia. As the disease is one of the upper motor neurones, a spastic element usually enters into the paralysis. It is met with typically in some cases of cerebral diplegia, in symmetrical cerebral sclerosis, in double hemiplegia, in progressive double hemiplegia from vascular disease, and in the “lacunaires,” where lacunæ of cerebral disintegration are found, more particularly in the basal ganglia. It is noteworthy that dysarthria may result from unilateral lesions in the path mentioned above, but the explanation of this is somewhat difficult. It may be, as has been held, that “the sudden cutting off of one-half of a bilateral innervation may temporarily, until the other half has had time to compensate for the loss, cause spastic paresis even in the most closely associated bilateral couple.”

If this were the sole explanation, dysarthria from unilateral lesions should occur more frequently than it does. Another somewhat curious point is that double hemiplegia does not always produce a complete anarthria, as *ex hypothesi* it might be expected to do. The probable explanation is that the lesions are rarely so placed as to interrupt completely all of the pyramidal fibers concerned.

The prognosis and treatment of dysarthria must be considered in the light of what has been said in an earlier part of the article on the prognosis and treatment of aphasia.

Prognosis.—The prognosis depends to a great extent on the diagnosis, in the sense that a diagnosis of the exact condition will determine the

physician in giving a favorable prognosis or the reverse. Excluding the dysarthrias of disseminated sclerosis, Friedreich's disease, general paralysis of the insane, and the other diseases enumerated above (for a consideration of which the reader is referred elsewhere), there is a group of transient dysarthrias comparable to the transient aphasias already noted. In unilateral cerebral lesions the dysarthria is often quite transient; whether it is more apt to be permanent in lesions of the left hemisphere than in lesions of the right (in right-handed persons) is a moot point. Obviously the position of the lesion, its nature and its degree or extent are of the first importance. The dysarthria of polioencephalitis may clear up just as in the cord cell-groups which have been thrown out of action by the local effects of the poliomyelitic toxin, but not actually destroyed, often recover. Similarly, in occlusion of the posterior inferior cerebellar artery, dysarthria produced by the ictus may be recovered from very soon, being less a direct than an indirect result of the lesion. Speaking generally, when the dysarthria ensues on vascular disease (endarteritis syphilitica, arteriosclerosis, embolism, and so on) the considerations mentioned on page 483 must be our guide in the matter. The age of the patient and the severity of the lesion are important factors in the situation.

At the best we may hope for comparatively little by way of "functional compensation," though what has been said will show that at the site of the damage a good deal of recovery may be attained by "functional restitution." Especially in pseudobulbar cases, where the lesion is bilateral, functional compensation is not a process from which help can be derived. For articulate speech the bulbar centres are in action simultaneously and equally, and if the lesion is bilateral nothing can be done. As far as the writer knows there is no evidence to suggest that extrapyramidal motor paths can be utilized to enable stimuli from the cortical centres to reach the bulbar nuclei by a circuitous route.

Treatment.—In the matter of treatment it is unnecessary to repeat what has already been dealt with fully in the section on aphasia. For dysarthria or anarthria, the physician must treat, on exactly the same lines as for aphasia, the underlying pathological condition, and, should it remain, its persistent clinical symptom-complex. All that requires to be said is that not infrequently cases of dysarthria prove amenable to reëducative handling. To quote Bastian: "The patient has to be assiduously drilled in the pronunciation of simple vowel sounds and consonants, at the same time that he is shown how to arrange the organs of articulation for the production of such sounds, whenever such instruction is needed. He is then schooled in the same way in the articulation of monosyllables, and subsequently in the combination of monosyllables into short words. A laborious process of tuition has, in fact, to be carried out from day to day—the patient has slowly to be taught anew how to speak." It is sometimes remarkable how much can be done for dysarthria by persistent drilling. Excellent results have been recorded by Haddon, Taylor, Wyllie, and others.

DYSPRAXIA AND APRAXIA

In the introduction to this article it was stated that apraxia is a subject which is as yet comparatively little known to the profession at large, so that a brief explanation of its nature and localizing value will not be out of place.

By dyspraxia or apraxia is signified the inability to perform certain purposive movements, in the absence, be it remarked, of either motor or sensory paralysis or ataxia. That is to say, the patient is unable on request to make certain simple movements volitionally, though his use of the same musculature automatically or involuntarily is a proof that there is no actual paralysis. Thus a hemiplegic may be unable to protrude his tongue when asked to do so, but he will put it out when taking a spoonful of marmalade. Again, he may be unable to use a pencil correctly with one hand, and yet fumble about with it in endlessly futile ways. He may employ it at once in the right fashion with the other hand. Apraxia may therefore be unilateral or bilateral. I have seen a patient with bilateral apraxia who is unable to clap her hands on request, although all the time she makes little fumbling movements with them in her eagerness to do what is wanted; performing the required movement in front of her does not help her. Apraxia may range to much more complex acts, *i. e.*, to more than mere isolated movements. A patient may perform simple movements quite well, but be lost in the carrying out of such a complex action as taking a match out of a matchbox and lighting a candle with it. Again, there may be a curious repetition or perseveration in the performance of certain volitional acts, so that the patient makes incorrect movements because he is unable to get away from a previous innervation.

It must be clearly understood that in diagnosing apraxia the physician must satisfy himself that the patient fully understands what is required of him; the latter must not have either auditory or visual agnosia, in other words; for in such a case his inability to perform certain acts correctly is dependent on the agnosia; it is agnostic apraxia, and not true motor apraxia. Similarly there must not be any gross sensory defect in the innervated limb, for that also may of itself interfere with correct motor action. With these provisos, if a patient cannot make either simple or complex movements when he recognizes what is required of him, or recognizes the objects which he is to use, and when there is no complicating paretic or anesthetic factor, then he is suffering from dyspraxia or apraxia.

A good deal of evidence has been accumulating which goes to show that in the frontal lobes, in the area which corresponds to what is known histologically as the intermediate-precentral region, there is situated a centre or centres for the coördination of the series of consecutive muscular innervations requisite for the due performance of an act. Such a centre may be designated a eupraxic centre, and reference has already been made to the fact that Broca's area and the writing

centre are probably best regarded as parts of this eupraxic centre, which controls the movements of all the segments of the body. It is not necessary to enter on the evidence here; the reader should consult modern text-books on the subject. From data which I have been collecting for some years now, I have found that lesions of the frontal poles, of the orbital surface of the frontal lobes, and of the precentral gyrus, are not likely to be associated with apraxia, whereas lesions involving the middle parts of the outer surface of the frontal lobes, and lesions in the centrum ovale of the same lobes, especially such as implicate the corpus callosum, are likely to reveal themselves by dyspraxia as a clinical symptom. Apraxia is therefore of localizing value; lesions in the distribution of the anterior cerebral artery on one or other side may be expected to be accompanied by that phenomenon. Lesions of the corpus callosum, anterior or middle third, are also in the same category in this respect. Any lesion which isolates the sensomotorium from the rest of the cortex, whether the lesion be anterior or posterior to the central gyri, may have the same effect. In some cases disease of the temporosphenoidal lobes is associated with apraxia; this, however, is not so well established.

The motor apraxic resembles the motor aphasic closely; the latter cannot speak although his speech musculature is not paralyzed; the former cannot "act" although his arm or his leg is not paralyzed. By thus correlating the two conditions the physician will gain a wider view of these pathological states, and will be in a position better to appreciate what will now be said of the prognosis and treatment of apraxia.

It should be noted, perhaps, that in the function of eupraxia it is highly probable that in right-handed individuals the left hemisphere leads and controls the right; that is to say, the correct ordering of specialized movements of both arms, say, is controlled from the eupraxic centre of the left brain. The converse occurs, presumably, in left-handed persons. It thus is brought about that a lesion in the left hemisphere may occasion dyspraxia in the left hand, either by itself, or with apraxia of the right hand also. I have seen an excellent example of dyspraxia in a mandoline player, who developed a right hemiplegia which eventually cleared up entirely; there remained, however, loss of the fine specialized movements of the *left* fingers necessary for the manipulation of the strings.

Prognosis and Treatment.—Since the commonest causes of apraxia are cerebral vascular lesions and tumors, the reader is referred to the paragraphs on the prognosis and treatment of these conditions under the heading Aphasia. He should also remember that apraxia occurs in a number of mental states a consideration which is outside the scope of this article. In general paralysis, at a certain stage, in dementia præcox, senile and arteriopathic dementia, and so on, apraxia is often to be found, if it is looked for. Its treatment is that of the underlying pathological state.

Where apraxia is established, in a stationary form, as a sequel to

vascular or other lesions, treatment by reëducation may well be considered. As far as I am aware, there is no case on record of systematic effort to reëducate an apraxic patient, although *a priori* no reason exists why an essay should prove abortive. Treatment must consist in the slow and laborious retraining of the limb for such acts as are lost or defective. Collier suggests that the regular taking out and replacing of the pegs of a cribbage board or of the marbles of a solitaire board would be useful exercises of a simple order. Unfortunately, the teacher will meet with a difficulty here, at least in some cases. If he performs the wished-for movements in the presence of the patient, and requests the latter to imitate him, he may find that the patient is unable to profit by the teaching, for he is unable to imitate. It is a fact that apraxic patients often evince indifference to their mistakes, and show a somewhat surprising inability to profit by them. Thus I have seen a patient who, when asked to put his forefinger on his chin, put it on the top of his head without any hesitation, with a smile as of satisfaction, and who repeated the mistake a moment later, though he was shown what to do. When told to put his left hand on his right ear, he put his left forefinger into his left eye, and when asked if he had done what I wished he nodded in affirmation. There can be little doubt that the apraxic patient, as a rule, has less insight than the aphasic, and is less put out at the mistakes he commits; he does not seem to be able to learn. This *Nichtkorrektur*, as the Germans call it, has been alluded to by more than one writer on the subject. For this reason, therefore, there may be difficulty in reëducating the patient along the lines which have been successful in cases of asphasia. If, however, the complacency and acquiescence of the patient in his apraxic condition can be overcome, if he can be stimulated at least to attempt what he is unable to perform, some headway may be made in the hands of an enthusiastic and patient teacher. Each case must be treated on its merits. It is impossible to lay down any definite rules, but the application of the principles must be relegated to the individual who is responsible in a particular case.

Agraphia.—One of the varieties of apraxia is agraphia, and this is a common symptom. It is, however, a highly complex disturbance. The centre for graphic kinesthetic ideas may be stimulated from the auditory-image centre, the visual-image centre, the motor-speech centre, and so on. Hence there may be considerable disturbance of one or other receptive centre without abolition of the faculty of spontaneous writing. Agraphia may be either sensory or motor in origin; motor agraphia is a form of motor apraxia. Strictly speaking a patient is not agraphic if he is able to write, however imperfectly, with the left hand, the right being hemiplegic, and it is curious how often a hemiplegic patient is able to make very fair attempts at writing, sometimes even to write well, with the left hand, without any practice. In the treatment of agraphia he must be taught again just as the child is taught at school, slowly and laboriously. If the right hand is out of action permanently, Bastian thinks that a new writing centre in

the right hemisphere must be trained into functioning, and this he supposes will be effected by stimuli from the right visual centre. It is quite possible, however, that the lesion may be so placed in the left hemisphere that the left writing centre is cut off from its control over the corresponding innervation centres in the precentral gyrus of that side, and yet be still in connection *via* the corpus callosum with the corresponding areas in the right hemisphere. By some way such as this the rapid acquirement of graphic power with the left hand can best be explained.

Where the right hand is not permanently paralyzed, or not at all paretic, an excellent way of restraining the patient to write is by the simultaneous employment of both hands in performing simple writing exercises, such as letters, geometrical figures, etc. He may be taught to trace over figures and designs marked on paper. It is well known that bilateral movements of this kind can be readily accomplished with a little perseverance, and in this way one hand will as it were reinforce the other. To render such exercises possible the path from the writing centre *via* the corpus callosum to the right Rolandic area, must not have been interrupted. If the patient is agraphic because of word-blindness, it is very questionable whether he will recover the faculty of writing, at least until such time as the word-blindness clears up.

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CHAPTER XII

TREATMENT OF STUTTERING

By E. W. SCRIPTURE, M.D.

STUTTERING is the term applied to a certain peculiar form of speech. The variations are innumerable. We meet, for example, the man who upon wishing to say something, says, "why, why, why, why," and so on for fifteen to twenty times, whereupon he makes his statement quite correctly. Very many stutterers use such a "starter."

A famous musical author was often suddenly caught in the midst of a sentence by a cramp that rendered him speechless for a quarter or a half-minute. One lady found herself incapable of asking for transfers on the cars, and was in a constant condition of exasperation on account of the extra fares she had to pay needlessly. One man was obliged always to buy his railroad ticket to the next town beyond his home because he could not say the name of his home town. One girl was in constant exasperation at being obliged to drink vanilla ice-cream sodas because she could not ask for chocolate, which she wanted. One gentleman could never get the blue-points that he wanted because the combination "bl" was too hard for him. One young man appeared at the Speech Clinic with the forename of "Harry." After a while his real name was found to be "John." The new name had been adopted on one occasion when he was seeking employment. He always had difficulty with the first letter of his name. When asked for it on this occasion he hesitated. Thereupon the employer had said "hurry up." He could not say "John," but the word "hurry" made it easy for him to say "Harry," and he had borne this name ever since. This same young man once served as a bell-boy. When sent to a room he would knock at the door and then retreat ten steps. When the door was opened he could walk up and speak; if the door was suddenly opened without allowing him time for this maneuver, he would be speechless.

Of a hack-driver who came to the clinic, the incident is related that a stranger once engaged him to go to a certain hotel. After a while he turned around and said to the passenger, "You g-g-g-g get out here, and g-g-g go back two blocks to the hotel." "Why didn't you stop at the hotel?" asked the passenger. "B-b-because I tried to say, 'wh-who-whoa' there, and could'n't get it out until now." "But why don't you drive me back?" asked the passenger. "B-b-b-because I might not be able to say 'wh-wh-whoa at all,'" replied the driver.

Few persons realize how terrible life becomes to a stutterer. A normal person may get a mild idea of it by supposing that every time before

he speaks he is obliged to wink one eye or to open his mouth and yawn; the feeling of embarrassment and shame would soon overpower him. A stutterer is worse off; every time he tries to speak he is obliged to make a dunce of himself in such a way as to make other people want to laugh at him. One religious but stuttering lady finally demanded to be "cured or chloroformed." One boy often threw himself on the floor, begging his mother to tell him how to die. Another boy asked for a letter to his father, telling him to keep the other children from laughing at him. Many stutterers become so sensitive that they imagine everybody is constantly making fun of them. The life of a stutterer is usually so full of sorrow that it can hardly be said to be worth living.

A very great injustice to the stutterer is the widely spread notion that stuttering is a bad habit which is to be corrected by reproof, scolding, and punishment. The treatment is supposed to consist in a kind of schooling, the result depending on the diligence of the pupil. Lack of progress is attributed to inattention or laziness. Parents, friends, and teachers are always alert to test the patient's progress. Of course, all this simply makes the stutterer worse, turns a mild case into a severe one, and drives many a sufferer to despair.

Causes.—The most frequent cause of stuttering is a nervous shock. Ghosts and other practical jokes, and, with very small children, such terrifying experiences as are found at amusement resorts (scenic railways, fire scenes, etc.) are often the causes of fright from which the child never recovers. Severe falls are just as often the sources of the mental shock.

A boy of twelve relates that at the age of seven on several occasions in the daylight, he thought he heard footsteps of some one following him in the hall, whereas the noise was of his own footsteps; thereafter he began to stutter. He is still afraid to walk in the dark. A young man of seventeen relates that he began to stutter in reading at seven years because he knew that he would make mistakes before the class.

Most of the stutterers from shock show a general condition of nervous excitability in which the predominant element is an abnormal state of expectancy toward persons and events. The patient is often on the alert for what is going to happen. He watches other people and replies before they half finish their remarks; or he is timid to such a degree that conversation is painful. The same condition of general over-anxiety I have found in patients who do not stutter. It is a typical psychoneurosis that may, perhaps, be appropriately called a "general anxiety neurosis."

A very frequent cause of stuttering is mental contagion by intentional or unintentional imitation. A boy thinks it fun to mock a stutterer, and ultimately finds that he himself cannot stop stuttering. Stuttering frequently appears after whooping cough, also after scarlet fever, measles, intestinal troubles, etc. The cause seems to lie in the condition of exhaustion.

Symptoms.—The most striking symptoms are cramps and spasms of the muscles connected with speech. Abdominal cramps are nearly

always present. Cramps and spasms of the muscles of enunciation are usually the most apparent ones.

A marked symptom is a cramp of the muscles of the larynx that reveals itself in a monotonous, hard, and often husky laryngeal tone. In the thousands of stutterers that I have examined this symptom was never once absent, even on occasions when there was no other evidence whatever that the patient was a stutterer. I have often risked a diagnosis of stuttering on this symptom alone; the procedure is useful on the not infrequent cases when the patient in my presence apparently does not stutter. It seems very strange that this symptom has never been noticed by any past writer on stuttering.

Other symptoms are shown in the excessive rapidity of speech, in the superfluous sounds or grunts that the stutterer may use to start himself, in the contortions of the muscles of the face or other parts of the body, in the huskiness of the tone, etc.

A never failing symptom is the lack of confidence of the patient in his ability to speak correctly. In many cases the thought, "Will I be able to say that word?" is sufficient to make it absolutely impossible for the person to say it. The stutterer always lives with the fear that his speech may "go back on him." Many a one is always thinking a few words ahead of what he is saying, being on the lookout for some word he thinks he cannot say. When such a word is coming, he avoids it by selecting another that will serve as well. One patient passed his life practically in always avoiding words; this mental work, being added to that of a normal man, kept him in a condition of nervous prostration.

The fear of being ridiculous is nearly always present. The person does not want to "make a dunce of himself." He therefore avoids reciting in school; he refuses invitations to social affairs; he would rather live with his father's employees in a mine than go to college; he shuts himself up and becomes a queer-mannered hermit, etc.

A condition of mental flurry is usually present. When the patient starts to speak, he becomes partly dazed by his emotion and does not know exactly what he wants to say. This condition may be present even when he does not stutter; in trying to answer a question, for example, he cannot make up his mind just what he wishes to say. Closely connected with this is a habit of hesitating in thought that sometimes arises. The mental flurry perhaps explains why some stutterers have most trouble whenever they are jocular. In some cases they stutter only when joking.

Stages.—Three forms or stages of stuttering may be distinguished. The first is that of mere habit that occurs when one child copies the stuttering of another. As long as the stuttering remains in this stage it will be dropped as soon as the cause is removed.

The stutterer nearly always goes beyond the habit stage. People laugh at him, mock him, scold him, threaten him with punishment, or whip him. Usually he is obliged to repeat words he stumbles on. He is made to go through exercises in reading and speaking. Extra hard words are given him to practice on. Speaking becomes a torture

for him. A new element, the "fear of displeasing and of appearing ridiculous," produces the "fright stage." The stuttering is now a distinct psychoneurosis that may have the most far-reaching consequences.

If the question is asked of a patient in the fright stage, "Why do you stutter?" he will answer, "Because I am afraid that I will stutter." Many a one will say that if he could only forget that he had stuttered, he would never stutter again. When the stutterer wishes to speak, the thought of his previous failures occurs to him and he feels or knows that he will appear ridiculous to those before whom he is speaking. This element disturbs his mental condition. He is seized with a violent emotion that may be described as stage fright before a single person. Embarrassment, shame, fear, etc., express themselves in his face and often disturb his mental actions so that he cannot think clearly. The emotion may make him absolutely speechless, as in the case of many patients who cannot say a word when introduced to strangers. Or it may make him stumble over his words; naturally he stumbles in the way he has learned to stumble, namely, with stuttering cramps.

The disturbance of mental action during the fright stage may produce a kind of intellectual paralysis. One patient was often unable to answer a question, not because he was afraid of stuttering, but because the requirement of answering actually paralyzed his mind so that he could not think of the answer. This habit had become so thoroughly formed in another patient that any excitement might render him unable to think; on the football field, where the system of signals required him to add numbers, he would, upon hearing the signals "six and four," which had to be added together, have to ask his neighbor how much they amounted to. One stutterer explained the mental paralysis when asked to give his name or any exact information as resulting from the fact that he is overwhelmed by having someone to depend on him for information that he alone can give.

A third stage occurs not infrequently. The stutterer is no longer embarrassed by his defect. It is obnoxious to him, and he would like to be rid of it, but the fright has disappeared. This may be termed "stage of indifference." It is usually found in older patients; they stutter because the habit is firmly fixed, not because they are embarrassed.

Analysis of the stutterer's condition of mind always shows a serious disturbance in his attitude toward other people. Most stutterers are shy and timid; the boldness or indifference in other cases is only a kind of bravado to cover up timidity. Much of this timidity is undoubtedly due to the effects of the stuttering, but its intensity is often out of all proportion to the occasion. It may well be that timidity is the basis on which stuttering arises. If this is true, stuttering would then be a condition in which timidity shows itself by a peculiarity in speech.

Social timidity shows itself in mental symptoms that are approximately the same in stutterers and non-stutterers; there are the same strained feelings toward other people, the same bashfulness, etc. The

bodily symptoms are also similar; the muscles of the body are more tense than they should be; there is often also the flushing of the face, etc. There are even resemblances in speech. The timid person who is a non-stutterer, speaks with a tense voice; he often stumbles over his words and sometimes can hardly get them out; he often sticks or reduplicates like a stutterer. If this timid "stuttery" speech can be supposed to be developed and firmly fixed in a set of habits, the result would be true stuttering.

The fact that stuttering arises only in some cases of timidity and not in others indicates that there is some other element in the disease. The following observations may perhaps suggest what it is. In several cases there has been a determined effort to get rid of the trouble and perfect good faith on the part of the patient, yet I have had the feeling that at the bottom of his soul the patient really did not wish to be cured. This reminds one of some forms of hysteria, psychasthenia, and neurasthenia, when the disease is really produced by the patient in order to obtain some end, although he is absolutely unconscious of this self-production. It may be suggested that stuttering is a defect which tends to exclude him from the society of his fellows, and that persons who really have this unconscious tendency instinctively seize upon such a means of encouraging it.

The same mental condition as that underlying stuttering is found in many cases of nervousness when quite other symptoms (headache, tremor, anxiety, etc.) appear instead of the speech trouble. It is often a cause of wonder why some neurotic patients are not stutterers. If we assume that the "isolation impulse" will use the most likely and effective means for its purpose, we understand why it seizes upon the speech function. We also understand that it will more readily disturb the speech when the mechanism of normal speech is less firmly fixed, as after exhausting diseases, fright or injury by imitation. When the normal mechanism is strong, the psychasthenic impulse must find some other outlet.

Stuttering is therefore a diseased state of mind, which arises from excessive timidity and shows itself in speech peculiarities tending toward a condition of segregation which will enable the person to avoid occasions where he will suffer on account of timidity.

Diagnosis.—In many cases the diagnosis of stuttering is apparently easy. It takes no great technical knowledge to declare a person a stutterer when he says his name is "T-t-t-t-Tommy;" it is as easy as to pronounce a man drunk when he is found lying collapsed and smelling strongly of alcohol; and the chances of making a mistake are just about the same. There are several conditions in which "stuttery" speech occurs without any of the pathology essential to true stuttering. I have known stuttering children to be brought to the clinic and diagnosticated as having hysterical mutism. I have myself made the mistake of diagnosticating as stuttering a case of "tic speech" occurring after chorea. I have also given the diagnosis of stuttering and begun the treatment in a case that was purely one of hysteria

with a peculiar speech movement. And I have diagnosticated and treated as lispers two cases of a peculiar trouble clearly allied to stuttering. The mistakes that can be readily made by anyone not an expert in speech defects may be imagined. It is related that one man was brought to Prof. Westphal's clinic with a diagnosis of a psychosis. When asked to speak, he would contort his face, make irregular movements with his body, and finally would run around in a rage. The trouble was simply that when he wanted to speak he could not produce the words. This made him so exasperated and angry that he acted like a madman.

The differential diagnosis of stuttering can be carried out satisfactorily only by a medical expert.

Hesitation in speech or repetition of words or syllables may arise with anyone in embarrassing situations, especially when the person is fatigued. The speech differs from that of a stutterer in not showing any cramps, and the condition is a temporary one.

The repetition of syllables in some nervous diseases, such as general paralysis, is due to an effort to hold a long and difficult word in mind. It is a mechanical repetition without any cramps. The repetition of words and syllables that occurs in some cases of motor aphasia is due to an effort to remember the following word or syllable; here again there are no cramps. The disturbance in the mental condition in these two conditions namely, organic nervous disease and aphasia, does not bear the slightest resemblance to that of true stuttering. There is no trace of the stutterer's embarrassment and fear. The failure of Kussmaul to distinguish between the syllable repetition and the word-repetition of these two conditions and the peculiar speech of stuttering, led him and later writers to see in stuttering merely a development of troubles of coördination. Such repetitions may be called "pseudo-stuttering."

Stammering.—"Stammering" is used in English in a sense identical with stuttering. The word should be avoided on account of the confusion with the German "stammeln," which means something entirely different, namely, defective enunciation arising from slovenly speech and from defects of the jaws and mouth.

Lisping.—"Lisping" is a term that may be applied to defective enunciation when this occurs as a speech defect. One of its forms may be termed "negligent lisping" or "slovenly speech." It is due to defective perception and execution of sounds. The condition is that of baby-talk which has not been corrected. Most frequently the child uses "t" instead of "s," or "t" instead of "th," saying "toap" instead of "soap," and "tick" instead of "thick," etc.

In "organic lisping" the defective enunciation is caused by abnormalities of the speech organs. An undershot jaw or a very high palate may produce a defective "s." Tongue-tie interferes with the "s."

Tic Speech.—"Tic speech" is characterized by spasmodic movements that break up the speech in a way somewhat like ordinary stuttering. The ordinary simple tic consists of a single movement which is

constant in character. The speech of the "post-choreatic neurosis" comprises a large number of tic movements. The trouble originates with acute chorea. After this has disappeared the spasmodic movements are retained as tics. A diagnosis of stuttering is usually made, but the trouble is of an entirely different nature. The stutterer may stick on a certain consonant; he nearly always has trouble in getting started. The tic speaker usually begins without trouble, but stumbles at any moment. There is no regularity in the sounds he has trouble with. The stutterer is usually very anxious and timid about speaking; the tic speaker is usually entirely unabashed by his defect.

Pseudo-stuttering.—Simple repetition of a word or of an initial syllable occurs to every one at times. It is frequently found in cases of neurasthenia, general paresis, motor aphasia, etc. Such repetition may be termed "pseudo-stuttering." The muscular movements are not cramp-like as in stuttering, and the symptoms are not of embarrassment and fear as in true stuttering. These peculiarities are usually sufficient to hinder a mistake in diagnosis.

Treatment.—The fundamental principle for treating a stutterer is to correct the defects that can be found in his speech, or in the mental or bodily functions connected with it.

The stiffness and monotony of the laryngeal tone is relieved by what might be called the "melody cure." "Melody" is the term used to indicate the rise and fall of the pitch of the voice. For singing it is indicated by notes on a staff. For speech it can be indicated by a line rising and falling.

The speech of the stutterer is monotonous. Its melody would resemble that of a straight line. Normal speech is very melodious.

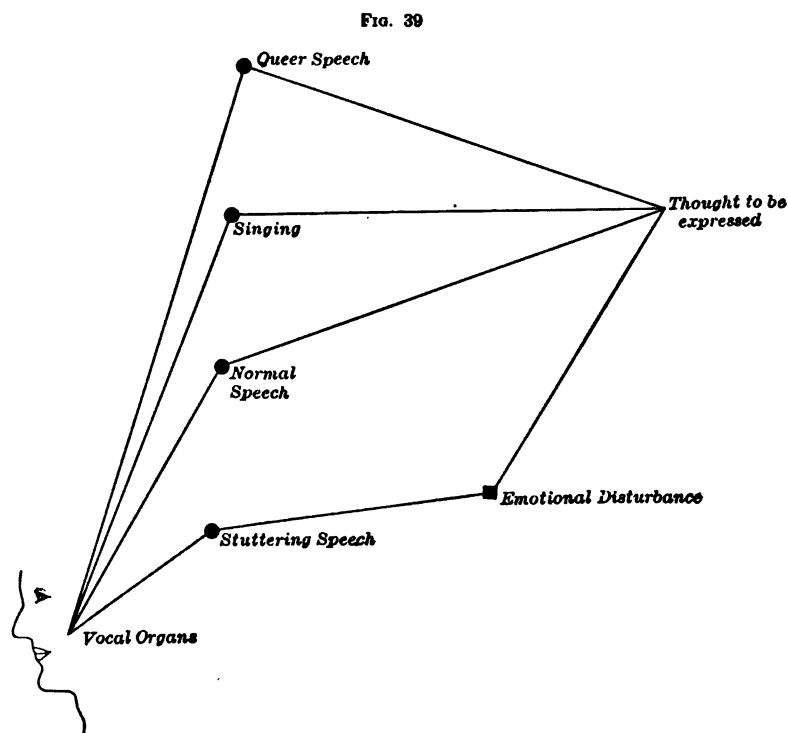
The pitch of the voice is regulated by the tension of the vocal cords. Constant pitch is not normal speech. The tension must be changed every instant, that is, the laryngeal muscles must be directly poised and must act readily and correctly. The stutterer cramps them up so tightly that they can move only with difficulty. He therefore sticks to one tone. To break up this cramp the stutterer is first taught to sing a song or phrase while accompanying the piano. His voice will rise and fall, and he will have no stiffness. He is then taught to speak the words of the same melody. This gives him the idea of melodious speech, in place of monotony. The next step is to make his voice flexible.

The psychological treatment of stuttering is based on the following principles:

The "principle of a new method of speaking" is founded on two facts: (1) that the stutterer always speaks in an abnormal voice, which we may call the "stutter voice;" (2) that he does not stutter when he expresses his ideas in any other voice, such as the singing voice.

The scheme shown in the illustration expresses these two facts: When the stutterer tries to express a thought in his usual way, the action of his speech is interfered with by the emotional condition—embarrassment or fear—that is aroused at the same time. He, there-

fore, speaks in his stutter voice. If he tries to express the thought in any other way than the usual one, the emotional disturbance does not arise. This explains the familiar fact that a stutterer never has any trouble when he sings what he wants to say.



Scheme to illustrate the mechanism of stuttering.

Since the patient does not stutter if he speaks in his usual way, he can be taught to speak in some kind of an odd voice. The stutterer can speak at any time without stuttering, if he will use an abnormally low voice, or an abnormally high one, or if he will drawl the vowels or slur the consonants, or again if he will speak in a choppy staccato voice, and so on. These are the methods of the "stammer-schools" and "stutter-curers." They are objectionable because they leave the patient with a queer voice; people often tell him that the cure is worse than the disease. The patient usually gives up the queer voice after a while and becomes a stutterer again because the queer voice itself produces embarrassment and he naturally feels like discarding it.

The essential point is that the stutterer should feel his acquired speaking voice to be different from his stuttering voice. One patient could never dictate to his stenographer. I found that he could not distinguish one note from another in music, so I told him to sing what

he wanted to dictate. He did so without the slightest hesitation or difficulty, in what he supposed to be a singing voice; it did not differ, however, from his usual voice, except in being slightly easier and more natural. As long as he thought he was singing he did not stutter, although he did not really sing. The cure was a failure because he refused "to make a dunce of himself by singing to his stenographer." To inform him of the fact that he did not sing would have destroyed the belief that he was singing and would have made him a stutterer again. There was no way out of the dilemma.

There is another way of speaking which is unusual to the stutterer, namely, the way in which normal persons speak. When he speaks in this way, he does not and cannot stutter. The therapeutic procedure on this principle will, therefore, be to teach him to speak normally. The various abnormalities will disappear when the patient has thoroughly learned the exercises below.

The "principle of habit-information" implies that the new way of speaking is to be drilled into the patient until it becomes a habit.

The "principle of spontaneity" is requisite because, when the patient has learned to repeat perfectly, he will still be unable to do so when he speaks of his own accord. A gradually increasing amount of spontaneous speech is introduced into his treatment.

The "principle of increasing embarrassment" arises from the fact that even when the patient has learned to speak perfectly in the presence of the physician or the instructor, he is unable to do so under other circumstances. The patient is taught to speak properly before a few persons or before a class. Still more difficulty is introduced by making introductions, speaking over the telephone, buying in stores, reciting in school, etc. For the introduction exercise the stutterer practises at first privately and then with a gradually increasing number of strangers.

The "principle of correct thinking" indicates that the abnormal habits of thought, which a stutterer always acquires to a greater or less degree, are to be corrected by appropriate exercises. A frequent abnormality is that of getting into a daze at each effort to think. The patient finds that he cannot decide promptly. One patient on being asked "Which kind of dog do you like best?" hesitated and grunted, and finally said, "I really cannot say which I like best." He was cured by being obliged to give some kind of decision quickly, regardless of whether it was correct or not. The trouble was due to the mental flurry or daze that had become a habit. Another patient, when leaving a house, found himself unable to say "Good-bye" because some friends were waiting for him. The trouble arose from a conflict between the motive to hurry after the friends and the motive of not offending the host; this produced a mental daze that left the patient speechless.

Exercises.—The following exercises present an outline of treatment that may be abbreviated as desired. Each step of each exercise is to be repeated a number of times. The exercises are to be repeated as often as may be needed.

EXERCISE I.—*Breathing in Preparation for Speech.*—The object of this exercise is to develop consciousness of breathing and to form a habit. It also teaches the "octave twist."

(a) The patient raises the forearms front upward inhaling, and lowers them side downward exhaling. He repeats this and each of the following at least four times.

(b) He rises the arms side upward inhaling, and lowers them side downward singing "ah."

(c) Likewise with the other vowels.

(d) The patient raises the arms upward inhaling; lowers them side downward singing "ah" on middle C and upper C (over the octave).

(e) Likewise singing words of one syllable.

EXERCISE II.—*Flexibility.*—The object of this exercise is to break up the hard monotonous laryngeal tone. It alone makes the voice normal when it is skilfully done, because it is impossible to stutter and to use the octave twist at the same time.

(a) The patient sings the vowel "ah" on each note of the octave.

(b) He strikes the lowest note of the octave and then the highest, singing the vowel "ah" half on the lowest note, half on the highest.

(c) He sings the vowel continuously (portamento) up the octave. He should note the "fog-horn" effect, the "octave twist."

(d) He practises singing the different vowels up the octave in this way.

(e) He sings a series of words with long vowels, running up the octavo in the same way.

(f) The instructor speaks a word with the octave twist. The patient repeats it.

(g) Same with a sentence, putting the octave twist on the first important vowel.

(h) Same with a poem, putting the octave twist on the first important vowel in each line.

(i) Same with prose, putting the octave twist on the first important vowel in each phrase.

(j) The instructor speaks sentences and questions with the octave twist.

EXERCISE III.—*Readjusting the Vowels.*—The object is to teach the patient to weaken the consonants and to speak slowly.

(a) The patient speaks each of the vowels in a long-drawn-out tone, going over the octave, *e. g.*, "a-h, o-h, etc."

(b) He speaks a short sentence, omitting all the consonants but lengthening the vowels; for example, the sentence "A snow storm is here" will be spoken "A-o-or-i-ere."

(c) He repeats the same sentence, lengthening the vowels and putting in the consonants only faintly.

(d) He learns to read and converse in this way.

EXERCISE IV.—*Smoothness.*—The object of this exercise is to teach the patient to shorten the consonants, as these are the sounds that cause him difficulty. At the same time it teaches him to speak

smoothly and easily. These are factors in forming a voice that is both normal and new.

(a) Repeat sentences making every important vowel at least three times as long as usual. These vowels should be spoken with the octave twist. The words are to be linked together, that is, there are to be no pauses or interruptions. The method may be indicated in this way: "Thesu—nisshi—ningbri—ghtly."

(b) Questions and answers and short sentences are to be spoken and read likewise.

EXERCISE V.—*Slowness*.—With a metronome beating fifty-four times a minute, the instructor and patient read, repeat sentences, ask and answer questions, etc., putting one syllable on each beat. A breath is to be taken before each sentence. The first important vowel in each phrase is to have the octave twist as described in Exercise II. The whole sentence must be spoken smoothly and softly without the slightest jerkiness. The object is to force the patient to speak slowly.

EXERCISE VI.—*Regulation of Breath in Speaking*.—This exercise is needed only when the patient has difficulty in breathing before each sentence.

The instructor and the patient are to hold sticks. Each time before speaking, the stick is to be raised sharply while breath is taken vigorously. After waiting one second with bated breath the person speaks.

(a) The instructor speaks a short sentence; the patient repeats it.

(b) The instructor asks a question; the patient repeats it.

(c) The instructor assigns a topic on which the patient is to say something. The patient must raise the stick and draw a breath before each statement.

(d) The patient is required to give a description of some object in the room in a similar way.

EXERCISE VII.—*Starting a Sentence*.—This exercise is needed only when the patient has special difficulty in starting. The accompaniment is to be gradually eliminated.

(a) The patient sings short sentences, striking a note on the piano as the first syllable is sung. Instead of using the piano, he may strike a bell or a table or hit his knee or make a gesture as in beating time.

(b) He repeats the same sentences, with the same accompaniment in the same way, but singing only the first word.

(c) He speaks them with the same accompaniment on the first syllable.

(d) Question and answer are sung with accompaniment on the first syllable.

(e) As before, but only the first syllable is sung, the rest being spoken.

(f) As before, but all spoken.

(g) The patient tells a story, singing the first word of each sentence with the accompaniment.

(h) He tells a story without singing but accompanying each first syllable.

EXERCISE VIII.—*Developing Expression.*—During these exercises no correction of faults is to be made. The entire attention is to be concentrated on the correct expression.

(a) The instructor repeats some poem with expression; the patient repeats it line by line, exactly imitating the expression.

(b) The instructor states a certain fact in a very melodious and expressive way; the patient repeats it exactly.

(c) The instructor gives a question as in (b); the patient repeats it exactly.

(d) The instructor gives a question in a very melodious and expressive voice; the patient answers it by taking a few words from the question.

(e) The instructor gives a question as before; the patient answers freely, but with the same melody and expression as in the question.

(f) Poems and prose pieces are recited with proper expression.

(g) Dialogues are read with the proper change of expression for each character.

(h) Jokes are read and spoken with an effort to give the most effective expression.

EXERCISE IX.—*Confidence in Reading.*—(a) The instructor and the patient are to read poems together. Then they are to read alternate lines together and alone.

(b) Same with sentences.

(c) They are to read a prose speech together, but the instructor is to remain silent occasionally.

(d) The patient is to read a prose piece, but the instructor is to join in at the first intimation of difficulty.

(e) The patient and the instructor are to read sentences and questions from a "traveller's manual," sometimes together, sometimes alone.

(f) The instructor and the patient are to read parts in a drama, the instructor joining in whenever the patient has difficulty.

(g) The patient is to read a paragraph and then tell its contents in his own language.

EXERCISE X.—*Confidence in Speaking.*—(a) The patient calls out railroad stations with a megaphone; the voice must be clear and decided.

(b) Same without the megaphone.

(c) The patient makes geographical statements with and without a megaphone; for example, "The Atlantic Ocean is east of the United States."

(d) He makes historical statements likewise (that is, with and without the megaphone); for example, "George Washington was the first president of the United States."

(e) Question and answer likewise.

(f) The patient relates a story or an incident likewise.

(g) He makes a speech likewise.

(h) He takes part in a debate likewise.

(i) He takes part in a continuous story which is arranged as follows: One person tells a story which he makes up as he goes along; he suddenly

stops, and the next person immediately continues the story according to his own ideas; he, in turn, suddenly stops and the following person continues. This is kept up until the story reaches the first person.

(j) A topic is assigned; the patient prepares a short speech to make on it and delivers it in the presence of the instructor.

(k) Same in the presence of several people.

(l) An impromptu speech is made on a given topic in the presence of the instructor.

(m) Same in the presence of other people.

The number of the people is to be gradually increased until the patient feels ready to get up at any moment and make a short speech on any topic

EXERCISE XI.—*Confidence in Buying*.—(a) The patient with a number of objects before him is supposed to be a store-keeper; other people (or patients) go to the store, inquire about articles, discuss the prices and buy. This must all be done with proper attention to slowness and melody of speech.

(b) The patient takes the part of the buyer.

(c) The store is turned into a railroad ticket office with the patient alternately as ticket agent and as traveler. Various questions are asked concerning trains, accommodations, etc.

(d) The ticket office becomes the box office at the theatre; the questions are to include location and seats, exchange of tickets, etc.

(e) When the patient can do this perfectly, the instructor accompanies him on actual purchasing expeditions to the stores.

EXERCISE XII.—*Scenes from Life*.—(a) A group of people is supposed to be in some familiar situation; for example, eating at a restaurant, riding in an automobile, forming a box party at the theatre, etc. The instructor works out the situation by description, while all persons present, including the patient, make the appropriate remarks.

(b) Similar scenes are worked out, the patient taking the leading parts.

(c) The group of persons is supposed to represent a club, the instructor occupying the chair. Various members are to make motions and discuss them, officers are to be elected, etc.

(d) The patient is made chairman of the club.

EXERCISE XIII.—*Spontaneous Speech*.—The object of this exercise is to remove the daze—mental cramp—which many stutterers have when they begin to speak.

(a) The patient says some word referring to an object placed before him or pointed out; the word must have some application to or connection with the object. For example, concerning a book, he may say "large" referring to its size, or "black" referring to its color, or "read" referring to its use, or "table" referring to its position, or "yesterday" referring to something it reminded him of, etc.

(b) He makes a statement slowly and melodiously concerning some object placed before him or pointed out to him.

(c) He names the object he sees on one side of the room, proceeding systematically from left to right, and speaking slowly and melodiously.

(d) He describes an object placed in front of him, using single words and proceeding systematically; for example, if a telephone is placed before him he will first use words referring to its appearance, then to its use, then to its faults, then to its history, etc. He should always adopt some such system in selecting words.

(e) Same as (d), but complete sentences are to be used instead of single words.

(f) Short sentences are to be spoken concerning objects not seen but more or less familiar; for example, breakfast, a distant city, George Washington, Atlantic Ocean, etc.

(g) A more extended account is required concerning similar objects as in (f).

(h) Some or all of the preceding exercises are to be carried out in the presence of additional people.

(i) When this can be done perfectly, the patient is to be called on to make short speeches on any topics that may be given him.

(j) He is to make speeches on topics of his own choosing.

EXERCISE XIV.—*Thinking*.—The object is the same as in the preceding exercise.

(a) The name of an object is called out. The patient calls out the name of some other object that suggests itself to his mind. If he is in doubt what to say, he chooses some object that is often seen together with the one mentioned. For example, on hearing the word "horse" he replies "cart." This process is called "association of ideas." At first the patient is to associate deliberately, taking as much time as he wishes. He practises for several times with words like the following:

hand	foot	hair
shoe	glove	eye
coat	sock	motor

(b) On hearing each of the words just used, the patient makes a sentence about it. It does not matter what the sentence states.

(c) On hearing each of the words he makes a sentence defining it.

(d) On hearing each of the words, he states some fact about the object implying something regarding its location or its use, or something that preceded it, or caused it, or followed it, or resulted from it, or has some relation to it.

(e) Starting with any given word he lets the mind bring up a long series of thoughts. These thoughts should not revolve around the original word, but should pass away to other subjects. If necessary the rule may be adopted of obliging the mind to leave the original word within three associations.

EXERCISE XV.—*Telephoning*.—(a) The patient calls someone on a private telephone, using the system of his town as nearly as possible. He first calls Central and then speaks with the person desired. He is to speak slowly and melodiously.

(b) He takes the part of Central and then of the person called up.

(c) He repeats (a) and (b) in the presence of other people.

(d) He does some of the most difficult exercises over the private telephone with the instructor or some other person at the other end.

(e) Using the public telephone, the patient puts his finger on the switch so that when he takes the receiver off the hook the telephone is not connected with Central. Someone sitting beside him represents Central and the person to whom the patient wishes to speak. He carries out exercises as on the private line.

(f) With the instructor close beside to him, the patient calls up Central and then some friends; if he has the slightest hesitation the instructor will speak for him.

(g) When he succeeds perfectly in the preceding, he tries the telephone independently. The instructor is to criticize his successes. The fear of the telephone is very marked in most stutterers.

The foregoing theory of stuttering, the principles of treatment and the exercises, have been developed in connection with the work of the Speech Department in the Vanderbilt Clinic, New York City.

They have been described from time to time in the *Journal of the American Medical Association* and the *New York Medical Record*. The exercises in their present form and part of the theory presented in the text are from my book on *Stuttering and Lispings* (Macmillan).

CHAPTER XIII

TREATMENT OF DISEASES OF THE CRANIAL NERVES AND ORGANIC LESIONS OF THE SPINAL CORD

By COLIN K. RUSSEL, M.D.

IN considering the treatment of lesions of the cranial nerves one is in most cases extremely limited, owing in the first place to their anatomical relations, most of the cranial nerves being intracranially situated—and at least as far as those parts are concerned which are most frequently affected by disease, local treatment is excluded. One must then treat the constitutional disease of which the symptom may be only a part, or, where possible and advisable, surgical intervention must be resorted to.

In order to direct the treatment into proper channels the various cranial nerves will be taken up seriatim with a brief review of such of their anatomical relations and of the lesions which may cause disturbances in their functions as may be necessary for a consideration of the subject in hand. The reader will then be referred where necessary to the proper chapters for the treatment.

THE OLFACTORY OR FIRST NERVE

The sensory end organs of the olfactory nerves lie in the Schneiderian membrane on the upper and middle turbinated bones and on the septum. The axones from these cells passing through the cribriform plate of the ethmoid bone divide into terminal arborizations about the cells of the second relay in the olfactory bulb, thence these neurones convey sensations to the cortical centre, the uncinate gyrus.

Lesions affecting the sense of smell may then be variously situated. The ordinary acute rhinitis or any local irritation may produce a loss of smell by congestion and edema of the nasal mucous membrane. Arrest of the nasal secretion due to disease of the fifth nerve may produce the same symptom, and obstruction of the passage of air by polypi may naturally throw these cells out of commission. Fracture or disease of the ethmoid bone, basal meningitis, or tumors of the frontal lobes may cause an anosmia on one or both sides from pressure on the bulb or tract.

Disease in the neighborhood of the olfactory centre in the uncinate gyrus is frequently associated with peculiar attacks called by Hughlings Jackson "dreamy states," which are ushered in by an aura of smell, usually of an unpleasant nature. The functional activity of the centre

is frequently inhibited in hysteria. Loss of smell not infrequently follows severe attacks of influenza and is probably dependent rather on the condition of the mucous membrane than on any organic lesion of the olfactory apparatus.

It must be remembered in testing the sense of smell that the nasal mucous membrane is supplied by the fifth nerve as well as the first, and that substances must be properly chosen to stimulate the olfactory nerves alone. Such are, oil of peppermint, oil of cloves, and tincture of asafetida. Volatile substances, as ammonia, vinegar, etc., cause a sensation by irritating the mucous membrane and the terminal end-organs of the fifth nerve and not because they possess any odorous qualities.

Treatment.—The treatment of disturbances of smell must then of necessity be dependent on the nature of the underlying cause and the reader is referred to treatises on the nose and its diseases, and to the special chapters dealing with the surgical treatment of tumors of the brain and fracture of the skull.

THE OPTIC OR SECOND NERVE

The sensory end-organs of the optic nerve are situated in the retina, their axones on their way to the brain are gathered together in the optic nerve. In man the majority of these decussate in the chiasm and terminate in the basal ganglia of the opposite side of the brain. The fibers from the temporal side of the retina subserving vision in the relatively small nasal field do not decussate but run to the basal ganglia of the same side. The macular fibers which subserve central vision run to both sides, thus gaining representation in both hemispheres. The external geniculate body appears to be the most important of the basal ganglia as far as vision is concerned, in man at least; reflex fibers, however, also go to the corpora quadrigemina and the pulvinar of the optic thalamus. Thence the next relay of neurones extends in the optic radiations through the extreme posterior part of the internal capsule back to the visual cortex about the calcarine fissure.

Disturbances of vision may then be caused by lesions variously situated, and the character of these disturbances will depend to a large extent on the site of the lesion.

Diseases of the Retina.—**Vascular Diseases.**—Anemia of the optic disk and retina is found in severe general anemias, whether due to toxic conditions or repeated hemorrhages. Hyperemia may be caused by constitutional or local conditions. Among the former may be mentioned general toxic conditions, chlorosis, hypertrophied and over-acting heart, convulsive seizures; it is always evident in the earliest stages of choked disk. Among the local causes is eye-strain, especially when associated with hypermetropia and astigmatism. The treatment consists in the removal of the cause.

Embolism of the Retinal Artery.—Occurs in ulcerative or vegetative endocarditis, and while it is not a frequent complication, cases do occur associated with chorea. It is usually monocular. If the embolus be large

enough to block the central vessel sudden and complete blindness occurs. If the embolism be small enough to pass into a branch of the artery partial blindness occurs corresponding to that part of the retina supplied by the branch; this usually goes on to complete blindness. The absolutely sudden onset of blindness is characteristic. The ophthalmoscopic appearances are characteristic. The arteries are empty and look like small white threads, the veins small and the disk pale and edematous; sometimes small hemorrhages are seen. Atrophy of the optic disk and retina follows rapidly.

Treatment.—If the case is seen immediately, deep massage of the eyeball through the closed lids may be practised. The maintenance of pressure allows a column of blood to be collected behind the embolus, which, with the release of the pressure, is forced against the embolus and may succeed in driving it on into the most peripheral circulation or breaking it up and thus permit of its dissemination into the general circulation. Repeated paracentesis of the anterior chamber or iridectomy with the object of reducing the tension has always been unsuccessful.

The patient should be kept in a recumbent position with the head low—care being taken to avoid cardiac excitement for fear of further emboli being let loose into the circulation.

Thrombosis of the Retinal Artery.—May give rise to an ophthalmoscopic picture identical with that produced by embolism. The points of differential diagnosis clinically are, previous attacks of transient blindness or a simultaneous attack of blindness in the opposite eye and premonitory signs of faintness, giddiness, and headache.

Treatment.—The treatment will consist in stimulating if possible a feeble circulation. The local treatment suggested in the paragraph on embolism will also be in order.

Retinitis.—Inflammation of the retina causes disturbance of vision, particularly of the central acuity—limitations of the visual fields or scotomas. Ophthalmoscopically it is characterized by diffuse cloudiness, especially of the central portion—there may be associated a marked congestion or swelling of the optic disk obscuring its outlines, constituting a neuroretinitis. The retinal veins become engorged and tortuous, small hemorrhages may occur, and a whitish exudate soon develops along the course of the vessels. Inflammation of the retina is seldom a local affection, but in most cases is due to a constitutional disease. Syphilis, either acquired or chronic, may cause a chronic diffuse retinitis. This is often associated with, or in fact is probably secondary to, an inflammation of the choroid. Albuminuria of pregnancy, or due to an interstitial nephritis, either acute or chronic, is also a frequent cause, always of bad prognostic significance, especially so in the latter case, patients showing this condition seldom living more than eighteen months to two years. Diabetes often causes a very similar picture.

Treatment.—The treatment will of course consist primarily in most active measures against the constitutional disease. In pregnancy,

especially if early in the course, abortion must be seriously considered. Locally dry cupping or leeches to the temples may be used as an adjunct. Complete rest to the eyes and protection from strong light by darkened room or dark glasses is also necessary.

Choked Disk; Papillitis; Optic Neuritis.—In choked disk there is hyperemia and swelling of the papilla, so that the outlines are obscured, partially in the mild cases, totally in the more advanced. The actual swelling of the disk may measure anywhere from 2 to 6 diopters or even more. The arteries are small, the veins engorged and tortuous; the physiological cupping filled in. There is in the more advanced cases considerable exudate about the vessels. Small flame-like hemorrhages are more or less frequent in the neighborhood of the disk.

It is not necessary to take up the various theories as to the methods of causation of choked disk. Bordley and Cushing in their recent investigations came to the conclusion that choked disk is due to the mechanical distention of the sheath of Schwalbe by obstructed cerebrospinal fluid. Whether this is the true explanation, as seems most likely, or whether, as von Leber suggests, the optic neuritis is due to the presence of some supposed irritant in the fluid distending the nerve sheath, does not signify in this place. It is important to remember that an advanced degree of swelling may exist without any disturbance of acuity of vision. If the choked disk remain unrelieved, however, secondary atrophy and blindness eventually follow.

Choked disk occurs in about 90 per cent. of cases of increased intracranial pressure from any cause. While its absence does not exclude intracranial tumor, especially one of a very slowly growing nature, its presence is very strong confirmatory evidence of such a diagnosis. It is found also in cases of chlorosis and in women where there has been a suppression of the menstrual flow following exposure to cold it is said to occur. Acute rheumatism and the eruptive fevers are not infrequently causative agents.

Treatment.—The treatment will be to remove the cause. In the case of intracranial tumors, a decompression operation, or when possible removal of the tumor, will permit a rapid subsidence of the swelling. Or if the tumor be of a specific nature the administration of antisyphilitic remedies; as described in another chapter, will, as a rule, relieve the condition. It must be remembered that choked disk leads on to atrophy and blindness, and palliative measures should be instituted before this occurs. Optic neuritis associated with chlorosis responds quickly to the administration of iron, and that caused by acute rheumatism disappears under salicylates. Pilocarpine, by causing profuse sweating, may be of aid in eliminating toxins from the system.

Chronic Retrobulbar Neuritis; Toxic Amblyopia.—This disease consists of a true neuritis of the optic nerve, usually beginning in the macular fibers. Ophthalmoscopically one sees a slight congestion of the disk with a slight indistinctness of its outlines, and this condition may go on to atrophy. The etiological factors are: the abuse of tobacco or alcohol, usually both, but the absorption of other toxic substances,

such as lead, the methyl compounds, bisulphide of carbon, dinitrobenzol (used in making explosives), will also cause a similar condition. Cases have been reported following the inhalation, over long periods, of the fumes from methyl alcohol.

The patient complains of a blurring of vision like a mist covering all objects, especially in a bright light. Central vision is particularly affected, giving rise to a central scotoma more marked, at least in the early stages, for color than for form. In the later stages the loss of central vision becomes absolute and the size of the scotoma increases. The affection of vision is always binocular. The progress of the disease is slow, occupying weeks or months.

Treatment.—The treatment consists in putting a stop to the use of the poison in question; the administration of potassium iodide may aid in its elimination. Strychnine given hypodermically, dry cupping of the temples and the nitrites have all been recommended: Galvanic electricity and the high frequency current applied to the temples are said to stimulate the retinal elements to activity.

Acute Retrobulbar Neuritis.—Acute retrobulbar neuritis occurs in the course of disseminated sclerosis in about 40 per cent. of the cases. It is due simply to a lesion of the disease happening to strike the optic nerve. It rapidly goes on to a partial atrophy with pallor of the temporal side of the disk, seldom leading to complete blindness. There is, however, an impairment of the acuity of vision with a permanent central scotoma.

Treatment.—Dry cupping or leeches to the temples have been recommended; it is doubtful how much influence they will have. Pilocarpine sweats may aid in eliminating the toxic cause of the disease.

Optic Atrophy.—Atrophy of the optic nerve may be either a primary process or secondary to an optic neuritis. The ophthalmoscopic picture differs considerably, especially in recent cases, but in cases of longer standing the differentiation is sometimes difficult. In both cases the disk is white, perhaps more of a chalky whiteness in the primary form. Its outlines are always very distinct in the primary form, while they may be slightly clouded in recent cases secondary to a papillitis. The vessels, both veins and arteries, are small in the primary while in post-neuritic atrophy the arteries are small and the veins are enlarged and somewhat tortuous. But the most important differential feature is the appearance of the physiological cupping which has a filled-in appearance in the post-neuritic, while in the primary form the cupping appears hollowed out, showing the laminæ cribrosa. In the partial atrophy following retrobulbar neuritis the temporal side of the disk may alone show the pallor; the cupping, however, has a partly filled-in appearance.

With atrophy of the optic nerve there must necessarily always be impairment of acuity of vision depending in its degree on the completeness of the atrophy. Thus in that form consequent upon a retrobulbar neuritis complete blindness seldom supervenes; other forms of atrophy usually go on to absolute loss of sight.

Treatment.—The treatment in either case must be prophylactic which in the secondary form means of course the relief of the papillitis before atrophy has set in. Either by decompression in the case of increased intracranial pressure or by energetic measures in the case of constitutional disease.

Primary optic atrophy comes on in the course of *tabes dorsalis* and as one evidence of an abiotrophic condition is sometimes associated with various forms of *Friedreich's ataxia* and other degenerative conditions, or may occur as the only evidence, usually in such cases. I have a case in my clinic at the Royal Victoria Hospital, several members of whose family are similarly affected, the condition appearing at about the same time of life in each case. To prevent optic atrophy in *tabes* one must guard against any habitual undue strain of vision either from the use of the eyes in poor light or subjecting them to a strong glare of light over any prolonged period. I have seen optic atrophy develop in a man, the subject of syphilis of some years' standing, after a prolonged exposure to the glare of the sunlight on the snow, and *Edinger* reports similar cases. It is well then to advise, under such conditions, the use of smoked glasses, the correction of any errors of refraction, and to strive in every way to prevent exhaustion of the optic nerve neurones. In the abiotrophic conditions the same advice would hold good. X-ray treatment applied to the temples might stimulate temporarily the retinal elements, but it could only be of the nature of a whip to the tired horse and the effect would soon disappear. Once the atrophy has developed it is beyond our power to alleviate the condition.

It is only necessary to mention the results of lesions of various parts of the optic nerve and tract in order to direct the reader to the proper chapter for their consideration from the point of view of treatment. A lesion of one optic nerve will cause blindness of the corresponding eye. If the lesion be in the chiasm it will produce a *bitemporal hemianopsia*, and if in the tract on one side a *homonymous hemianopsia*, but as fibers from the macular regions of either eye are represented in both tracts central vision will be spared. The optic nerve, as has been mentioned, contains also reflex fibers. The reflex arc, with the third and sympathetic nerves, governing the movement of the pupil, is probably by way of the region of the *corpora quadrigemina* so that with a lesion of the optic nerve causing blindness, if light be thrown into the pupil of the blind eye the pupil does not react; whereas if light be thrown into the good eye the pupil of the blind eye contracts perfectly. With a lesion of the optic tract, besides the *hemianopsia* above mentioned, there is also an interference with these reflex fibers so that if a ray of light be thrown on the blind side of the retina no reaction of the pupil takes place. This must be done with care as of course any stimulation of the healthy side of the retina will cause a reaction of the pupil. Such a *hemiopic pupillary inaction* denotes a lesion in the optic tract or basal ganglia and will not be present if the lesion be in the optic radiation or occipital cortex.

OCULAR MOTOR OR THIRD, FOURTH, AND SIXTH CRANIAL NERVES

The motor nerves of the eye are: (1) The third or oculomotor, which supplies the levator palpebræ, the ciliary and constrictor of the pupil, superior internal and inferior recti, and inferior oblique. (2) The fourth or trochlearis supplies the superior oblique. (3) The sixth or abducens supplies the external rectus. (4) The sympathetic coming from the cavernous plexus of sympathetic nerves originates in the cervical cord and supplies the dilator pupillæ, Müller's muscle and the vessels of the orbit.

Anatomical Relations.—In order to take up the treatment of lesions of these nerves rationally and to direct therapeutic procedures along proper avenues, we must refer briefly to their anatomical and physiological relations.

Third Nerve.—The third nerve, taking its origin from groups of cells in the floor of the aqueduct of Sylvius, sends its fibers in several small bundles through the crus cerebri; these pass through the red nucleus on their way, and leaving the brain at the anterior edge of the pons, where they are gathered together into one bundle, pass along the outer wall of the cavernous sinus and enter the orbit through the sphenoidal fissure. It would appear that each of these groups of cells forming the nucleus represents an individual muscle so that lesions in the neighborhood of the nucleus or the bundles of fibers as they pass through the tegmentum of the pons may pick out only certain groups causing a paralysis of only some of the individual muscles supplied by the nerve. Lesions of cerebral peduncle involving the third nerve will therefore cause paralysis of the ocular muscles on the side of the lesion, and by the destruction of the pyramidal tracts before their crossing will therefore give rise to a spastic paralysis of the limbs of the opposite side—Weber's syndrome. (Plate XXXIX, Fig. 1.) On the other hand, lesions occurring after its exit from the pons are usually associated with disturbances in function of all the muscles supplied.

Fourth Nerve.—The fourth nerve, arising from a similar group of cells, lying a little more caudalward in the floor of the aqueduct, sends its fibers posteriorly where they decussate in the roof of the aqueduct of Sylvius at the level of the posterior corpora quadrigemina and come to the surface on the posterior surface of the superior cerebellar peduncle. It then winds around the crista of the midbrain and runs with the third nerve along the outer surface of the cavernous sinus, passing into the orbit through the sphenoidal fissure.

Sixth Nerve.—The sixth nerve arises from the floor of the fourth ventricle near the median line and runs in a few scattered bundles in an anterior direction through the lower part of the pons, making its exit in the groove between the pons and medulla. Its course thence to the sphenoidal fissure, where it enters the orbit, is almost directly forward and in many cases at least it underlies the lateral branches of the basilar artery.

PLATE XXXIX

Fig. 1



Weber's Syndrome. Third Nerve Paralysis on One Side and Hemiparesis of the Extremities of the Other.

Fig. 2



Third Nerve Paralysis. Ptosis and External Strabismus.

Fig. 3



Paralysis of the Fourth Nerve. Inability to look down. It will be noted that as the eyeball does not turn downward the eyelid does not descend, at the same time the patient can close his eyes when told to do so.

Fig. 4



Paralysis of the Fourth Nerve. The same patient closing his eyes to command.

The nuclei of these nerves are all in close touch with each other and with other nuclei, especially Deiters' nucleus of the eighth nerve and with the facial by means of association fibers which run in the posterior longitudinal bundle.

Ocular Paralysis.—Paralysis of the third nerve will give rise to ptosis or drooping of the lid with dilatation of the pupil and a divergence of the eyeball outward and in a slightly downward direction owing to the unopposed action of the external rectus and superior oblique. (Plate XXXIX, Fig. 2.) On the patient attempting to look at any object there will be noticed a contraction of the occipitofrontalis and an elevation of the eyebrow in an attempt to overcome the ptosis. The patient will complain of double vision and frequently of vertigo and nausea on attempting to use the eyes. On examination there is also inability to move the eye in an inward, upward, or downward direction.

Paralysis of the fourth nerve causes slight impairment in downward movement of the eye, and owing to the uncontrolled action of the inferior rectus the eyeball turns inward slightly. (Plate XXXIX, Figs. 3 and 4.) This lesion gives rise to diplopia in the lower half of the fields, usually most noticed in going down stairs.

Lesions of the sixth nerve give rise to a paralysis of outward movement. The eyeball, owing to the unopposed action of the internal rectus, turns inward, causing diplopia. (Plate XL, Fig. 1.)

Lesions of the cervical sympathetic, by paralyzing Müller's muscle, cause a retraction of the eyeball with narrowing of the palpebral fissure. The pupil, owing to the action of the unopposed sphincter pupillæ, is contracted.

The normal pupil reacts to light and to accommodation, but under certain circumstances disturbances of these reactions occur. Some have already been mentioned, but the most common is known as the Argyll-Robertson pupil. In this the pupil retains its reaction to accommodation but does not react to light. This condition is practically pathognomonic of syphilis of the nervous system and is due to a break somewhere in the reflex arc. The exact mechanism of its production is not of importance here as there is no known method of affecting it by treatment, but its presence should direct one's attention to the underlying constitutional disease and one's efforts into the proper channels.

Of the paralyzes affecting the external ocular muscles that of the external rectus is by far the most frequent. The long and direct course of the sixth nerve exposes it to injury to a greater extent than is the third nerve. The fourth nerve is seldom affected. The same causes which produce oculomotor paralysis may cause sixth nerve paralysis. Thus basal meningitis, syphilitic, tuberculous, or simple, may affect any or all of these nerves; tumors of the base likewise may involve any one of them. Abducens paralysis also occurs frequently associated with conditions of increased intracranial pressure without having any localizing significance. This is explained by Collier as follows: The course of the sixth nerve, from its exit from the pons to the sphenoidal

fissure, is directly forward. A cerebral neoplasm, in order to make room for its growth, has a tendency to force the brain back through the only opening there is, namely, the foramen magnum. Thus a backward pull on the sixth nerve is set up, usually more on one side than the other, and a paresis follows. Cushing, on the other hand, believes the frequent involvement of the sixth nerve in these cases is due to pressure on the nerve by the lateral branches of the basilar artery rendered tense and hard by the arterial hypertension which may be secondary to the increased intracranial pressure.

Fractures of the base of the cranium may also involve these nerves. Vascular lesions of the pons, either thrombosis or hemorrhage, involve the third more frequently than the others, and by affecting one group of cells of the nucleus, or one bundle of fibers, may cause paralysis of certain members only of the group of muscles supplied by it. In recent epidemics of acute poliomyelitis many cases have occurred in which a condition of polioencephalitis superior involving the nuclei of these nerves gave rise to a paralysis more or less permanent. Among the etiological factors less serious as far as the ocular paralysis is concerned, are, multiple sclerosis, diphtheria, ptomain poisoning, influenza, and exposure to cold.

Treatment of Ocular Paralysis.—In all cases the first essential is to remove the cause; in the case of an intracranial neoplasm either the removal of the tumor, when this is possible, or a simple decompressive operation, if done early enough, will quickly relieve an abducens paralysis which has not been caused by actual involvement of the nerve in the growth. In the case of an underlying constitutional disease, as tabes and syphilis, the treatment is described elsewhere. In acute cases the result is usually good. It is only necessary here to mention the direct symptomatic treatment. Gowers recommends the employment of counterirritants if the onset be acute or subacute, or if the other symptoms suggest inflammation; a blister being placed behind the ear or at the occiput when the disease is probably at the base of the brain, on the temple if it is in the orbit. This treatment is often followed by a striking increase in the power of the affected muscle. Salicylates and diaphoretics are indicated in rheumatic cases and those following exposure to cold, and hot fomentations also should be applied to the forehead and about the orbit. In most cases of local neuritis mercury is also indicated, even when there is no indication that the condition is due to syphilis, it being a most efficient agent. Ocular palsies resulting from diphtheria, influenza, multiple sclerosis, and ptomain poisoning are, as a rule, not permanent; the latter especially are of short duration. Tonic treatment is indicated. Direct treatment with electricity is impossible; even under cocaine the muscles cannot be satisfactorily stimulated, and repeated treatments would very soon endanger the eye. The galvanic current would be the only one of any use, but to give it in sufficient strength to stimulate the muscle to action is quite impractical.

The diplopia due to the partial paralysis of a muscle may be relieved

by exercises in following a moving object with the eye or by the use of a prism, which should never be quite strong enough to completely correct the diplopia, as that would do away with all voluntary effort on the part of the paralyzed muscle. The prism to be beneficial should just be strong enough to permit fusion of the images by muscular action, and should be used for an hour or so each day. Such gymnastic exercises often give very satisfactory results.

If the paralysis is stationary and has remained so for many months one might relieve the condition by the advancement of the tendon of the paretic muscle if the paralysis is only partial, but apart from this, operative interference is not advisable, save only when contractures have developed in the antagonistic muscle and the paralyzed muscle has regained power but cannot work against the shortening of its opponent.

The suggestion of the advancement of another muscle in the same eye, as, for example, the superior rectus where the inferior oblique is paralyzed, does not seem rational. The action of these two muscles is not identical and cannot be made so, and to bring the images closer together than formerly, as a result of such advancement, is not to relieve the condition one iota. Tenotomy of an associate muscle in the fellow eye, as, for instance, the external rectus of one eye when the internal rectus of the other is paralyzed, and so on, is said to give very good results, but most patients will probably prefer to have one normal eye and cover the other with a patch or ground glass.

Nystagmus.—Is the name given to the condition in which the eyes are affected by peculiar rhythmical, jerky movements usually bilateral and similar in each eye, produced by alternating contractions of opposing muscles. To be pathological it must be more than a mere tremor. Nystagmus occurs normally under certain conditions, as when one looks out of the window of a rapidly moving train at the nearby scenery; pathologically it is caused by local affections of the eye which interfere with sight. It is frequently congenital, usually associated with very marked errors of refraction. It occurs also in miners and apparently is due to their occupation—working in the dark in cramped positions. It does not occur nearly so frequently in well-lighted mines. It occurs also in disseminated sclerosis, Friedreich's ataxia, and especially in lesions of the cerebellum.

The movement is practically always bilateral—usually horizontal, but sometimes vertical or rotary. It is sometimes associated with a slight movement of the head corresponding to that of the eyes in time and direction.

Treatment.—The treatment is to remedy when possible any defect of vision. Miners should be advised to change their occupation, or at least they should have better light at their work. When associated with a lesion of the cerebellum it may be an aid in localization, but in itself will not call for special treatment.

The Trigeminal or Fifth Nerve.—Anatomical Relations.—The trigeminal or fifth nerve is the great sensory nerve of the face and motor

nerve to the muscles of mastication. The sensory portion corresponds to the posterior sensory roots of the spinal cord and is divided into three branches.

1. **The Ophthalmic Nerve or Superior Branch.**—The ophthalmic or superior branch, making its exit into the orbit through the sphenoidal fissure, supplies the skin over the upper eyelid and upper part of the nose, and passing through the supra-orbital foramina supplies the forehead and front part of the head; it supplies also the tear ducts and glands. Paralysis of this branch gives rise to loss of sensibility in the region supplied and to dryness of the eye.

2. **The Superior Maxillary or Middle Division.**—The superior maxillary or middle division sends a branch to the meninges. It then makes its exit from the skull through the foramen rotundum, supplies the lower eyelid, cheek, and side of the nose, the nasal mucous membrane, the upper lip, and the upper jaw with the teeth. Paralysis causes loss of sensation over these parts and dryness of the nasal mucous membrane.

3. **The Inferior Maxillary or Inferior Branch.**—The inferior maxillary, or inferior branch exits from the skull through the foramen ovale and supplies a part of the meninges, the lower part of the cheek about the mouth, the lower lip, chin, lower teeth and gums, part of the mucous membrane of the mouth and of the salivary glands. Paralysis will cause loss of sensation over the above parts and lessened secretion from the salivary glands on this side.

The cells of origin of the trigeminal nerve are in the Gasserian ganglion, which, lying in a depression in the middle fossa of the cranial cavity near the apex of the petrous portion of the temporal bone, corresponds to the posterior ganglia of the spinal sensory roots. Its inferior division after leaving the ganglion and emerging from the foramen ovale joins—like the spinal roots—with the so-called motor branch of the fifth nerve. From this point on it is a mixed nerve, the motor fibers supplying the pterygoids, the masseters, and temporal muscles—the muscles of mastication. A lesion of the motor branch of the nerve is easily recognized clinically if the fingers be placed on the masseters or temporal muscles and the patient be asked to close the jaws forcibly and equally. The lessened amount of contraction or the absence of it in the affected muscles is obvious. If the mouth be widely opened the jaw will deviate to the affected side owing to the paralysis of the pterygoid muscles. (Plate XL, Fig. 2.) The lingual branch of the division supplies the mucous membrane of the tongue and conveys in its sheath fibers from the taste papillæ of the anterior two-thirds of the tongue; these taste fibers leave the lingual nerve in the chorda tympani which joins with the seventh nerve and probably conveys these fibers to the brain in the pars intermedia of that nerve, ending in the fasciculus solitarius along with fibers of the glossopharyngeal nerve from the taste papillæ in the posterior third of the tongue.

The proximal root of the Gasserian ganglion enters from the side of the pons nearer its upper than its lower border and terminates in an

PLATE XL

Fig. 1



External Rectus Palsy due to a Lesion of the Sixth Nerve.

Fig. 2



Paralysis of Both the Motor and Sensory Fifth. Trophic Ulcer of the Right Eye, and Deviation of the Jaw to the Paralyzed Side on Opening the Mouth.

ascending and a descending root about cells in the substantia gelatinosa, corresponding to the termination of the posterior spinal roots. The motor root takes origin from a nucleus just internal to the substantia gelatinosa at the level of exit of the nerve. It has been necessary to go into the anatomical relations and the physiological functions of the nerve in some detail as modern treatment of some of its lesions requires this knowledge.

Etiology.—Lesions causing disturbance of function of the fifth nerve may be (1) in the pons—and may be either of vascular origin—whether hemorrhage or softening, or from involvement of tumor growth, foci of sclerosis in multiple sclerosis, or foci of inflammation in poliomyelitis. In these cases either the nucleus of the nerve or its fibers may be involved.

2. At the base of the brain the nerve root may be involved by syphilitic meningitis, tumors, or from hemorrhage secondary to a fracture of the base either in the middle or posterior fossa.

3. Each branch of the nerve may be damaged by special lesions, growths of the pituitary gland, or an aneurysm of the carotid may affect the superior branch, and tumors of the sphenomaxillary fossa may involve the second and third divisions. Primary neuritis of the fifth nerve is relatively rare; gout and syphilis may be predisposing causes.

Paralytic Conditions of Fifth Nerve.—Symptoms.—The symptoms of paralysis of the nerve have been discussed under the respective branches. Neuralgic pains often precede the paralysis, when the lesion is caused by the irritation of a new growth, the duration of this irritative stage will of course depend on the rapidity of growth and involvement of the nerve.

Treatment.—The treatment of paralytic conditions of the fifth nerve will of course depend on the cause, but much can be done to alleviate suffering from the local condition produced. For example, when the superior branch is involved, great care should be taken of the eye; the lacrymal secretions may be much lessened, and besides the consequent dryness there is a tendency to ulceration of the cornea from the loss of its trophic nerve supply, and as the conjunctiva will be insensitive the patient has no knowledge of the presence of small foreign bodies on its surface. It is necessary, then, not only to protect the eye against the entrance of particles of dust and so forth, but by means of simple lotions, *e. g.*, acid. boric., sod. bibor., āā gr. vj (0.4); aquæ camphoræ, ʒj (30.0), to keep the conjunctiva frequently moistened and to wash out any foreign particles that may have gained access. Gowers recommends the occasional application of a drop of castor oil to the conjunctiva to aid the subsidence of a beginning infection. The success of all such measures, however, as he points out, will depend on the degree of irritation of the nerve. When intense the rapid inflammation baffles every attempt to arrest it, and one must beware of a secondary infection of the other eye.

When the middle and inferior branches are affected with loss of

sensibility in the mucous membrane of the cheek and in the side of the tongue a cleansing mouth wash is very necessary to remove the furring that collects from disuse of the insensitive side of the mouth in chewing. The inside of the cheek when insensitive may be inadvertently bitten and such wounds are slow to heal owing to the loss of trophic impulses.

Neuralgia in Region of Fifth Nerve.—Neuralgia in the region of the fifth nerve, facial neuralgia, and trigeminal neuralgia are synonymous. The term *tic douloureux* is reserved for a particular variety of the disease.

Etiology.—Facial neuralgia is a not uncommon affliction, especially in adult life. It is usually seen in neurotic individuals; gout and rheumatism are often predisposing factors, chlorosis also, and in general any morbid cause capable of enfeebling the organism. It frequently follows exposure to cold. Chronic toxic influences, as in poisoning by alcohol, lead, and malaria, are sometimes of etiological importance, as are also some of the acute infections, as influenza.

Local causes capable of producing facial neuralgia are numerous. Disease of the meninges—it may be syphilitic—disease or injury of the base of the skull or of the bones of the face in the neighborhood of the branches of the nerve, may by involving or compressing it, produce neuralgia. Its long course through bony canals and the superficial position of the terminal branches expose it to many and various sources of injury and disease. Dental caries, alveolar abscess, inflammation of the mucous membrane linings of the various air spaces, all may set up severe pain in the region of the branch of the fifth nerve supplying the part, with not infrequently referred pain in the regions of the other branches. The pains of tabes occasionally occur in the territory of the fifth nerve, probably secondary to an inflammation.

Symptoms.—Pain is the essential and often the only symptom of facial neuralgia. Preceded sometimes by a prodromal paresthesia this pain may be mild at the onset and gradually become accentuated or it may start in at its maximum intensity. It is rare, save in syphilitic cases, that both sides of the face are affected, and only exceptionally are all branches of the nerve on one side the seat of pain. In such cases the pain is usually fairly continuous, often not very severe but insupportable on account of its persistence.

Tic douloureux, as a rule, affects older people and is often associated with marked arteriosclerosis. It comes on in paroxysms often of extreme intensity, lasting, as a rule, only a moment, but repeated with great frequency; starting from one point and radiating to the periphery of that branch, it often passes over to other branches. Any movement of the face in speaking or eating, any draught of air, or any touch may be sufficient to bring on the pain. Immediately there is a reflex spasm of the muscles of the face, the skin flushes, the eye may become injected, the secretions from the lacrymal glands and nasal mucous membrane are usually increased, and the skin is left exceedingly hyperesthetic, especially, as Valliere observed, over the points where the nerves pass

out of the bony canals to lie superficially. This condition of affairs may last anywhere from a few days to many years, with more or less short intervals of comfort, if it might be so described, when the patient has the constant fear of a recurrence of the pain hanging over him, although some patients do get well spontaneously.

Pathology.—As a rule there does not appear to be much evidence of pathological changes to be found in the nerve or its ganglion; sometimes a sclerotic condition of the ganglion betokens a former low grade of inflammation, in other cases Dana has found an obliterating end-arteritis of the vessels of the nerve itself, causing defective nutrition of the nerve accompanied by vascular spasm.

Diagnosis.—Diagnosis is, as a rule, not difficult; in the symptomatic group the local cause is, as a rule, obvious, and one must direct one's energies against it; especially in syphilis and malaria is treatment rapidly efficacious.

Treatment.—To relieve the acute pain a counterirritant paint of equal parts of chloral hydrate and camphor, painted on carefully so as not to spread on the mucous surfaces nor to encroach on the border of the hair, as it has a tendency to turn this gray, is often helpful, especially if combined with good doses of aspirin and phenacetine. Counterirritation with a faradic current applied by means of a wire brush sometimes does well; but perhaps more effectual is the application of a weak constant current, the anode being placed over the painful spot and the cathode on some indifferent part of the body. The current should be turned on gradually after the anode is placed on the part, and after being maintained at about 5 to 10 milliampères for several minutes, gradually diminished again before the electrode is removed. In a large number of cases occurring in early and middle life the trouble seems to rest on a rheumatic basis; in these the salicylates may be used energetically. Gelsemium in full doses, beginning with 10 minims of the tincture and gradually increasing, or cannabis indica in doses of $\frac{1}{4}$ to $\frac{1}{2}$ a grain of the freshly prepared drug, will sometimes control the pain. Strychnine given in ascending doses, beginning with $\frac{1}{80}$ grain three times a day and increasing gradually until the physiological limit of the drug is reached, has been recommended. Associated with the above measures the general health should be attended to, and for this purpose cod-liver oil or iron tonics are often necessary.

In the pain from dental caries, when a specialist's aid is not immediately possible, a triturate of atropine placed in the mouth in the neighborhood of the offending tooth and allowed to dissolve, and the solution allowed to lie in the mouth about the tooth for a time, will often give temporary relief.

In the treatment of tic douloureux all these measures may be tried and may be combined with the various forms of suggestion and even hypnotism in suitable cases, though, as a rule, they are more or less ineffectual, except perhaps temporarily, and one is driven to resort to more radical means for the relief of the excruciating and insupportable pain. However, it is no longer such a serious matter as formerly when

the radical cure meant the extirpation of the Gasserian ganglion with all the attendant risks to life, which are especially great in many sufferers of advanced years and marked arteriosclerosis.

Injection of Alcohol.—The extirpation of the ganglion may still remain the only radical cure, but the injection of alcohol into the nerve branches at the point of their exit from the skull gives instantaneous and prolonged relief and can be repeated if necessary. It is, moreover, practically without danger in competent hands and the technique may be easily acquired by any properly trained man. The first requisite is absolutely strict asepsis. One should also have at one's disposal several skulls or autopsy material on which to practice and perfect the technique. All that is required in the way of instruments is: (1) A straight needle about 12 cm. long, 1.75 mm. thick, not acutely sharp, with a stylet, the somewhat rounded end of which is flush with the needle point when the stylet is pushed home. The needle is graduated in centimeters from the point up to 5 so that the operator may know just how deep he has penetrated. (2) A glass syringe holding about 2 c.c., with a smooth nozzle to fit this needle.

In operating a general anesthetic is seldom necessary nor is it advisable, as one must be dependent on the patient's sensations. Ethyl chloride may be used to freeze the skin where the needle is to be introduced. The stylet is partially withdrawn when the needle is pushed through the skin and subcutaneous tissues; it may then be pushed home, thus preventing the puncture of any large vessel. A burning pain referred to some part of its distribution announces to the operator that the needle has penetrated the nerve, then the stylet should be withdrawn, the syringe connected to the needle, and the injection made. This should be followed either immediately, or within a few minutes, by loss of sensibility to pain in the area supplied. It is well to leave the needle in place until it is ascertained if this be the case, after which the needle may be slowly withdrawn. Various combinations of alcohol are used by different operators; thus Patrick, of Chicago, finds the following satisfactory:

R—Cocain, muriat.	0.1
Alcohol	13.5
Aq. destil.	q. s. ad 15.5

Blair, of St. Louis, recommends

R—Novocain	2 per cent.
Chloroform	6 per cent.
Aq. destil.	22 per cent.
Alcohol	70 per cent.

Either is good, the quantity injected being about 2 c.c. for each nerve.

When the supra-orbital branch is affected the nerve may be reached by making the needle enter just below the external angular process of the frontal bone, and by following along the malar maxillary frontal

suture for a distance of 3 or 3.5 cm., where the outer exit of the sphenoid fissure is reached. It will, however, be remembered that the optic foramen is above the inner end of the fissure and the oculomotor nerves also pass through this fissure. Out of deference to these the supra-orbital branch should not be attempted by deep injection, but should be reached in the supra-orbital foramen, the injection being introduced as deeply as possible. In treating this branch, if using Blair's formula, it is well to leave out the chloroform or reduce it to 2 instead of 6 per cent.

In the case of the superior maxillary or middle branch the needle should be introduced a little below the zygoma and passing through the sigmoid notch of the mandible the pterygoid process is encountered and its junction with the great wing of the sphenoid recognized; by working forward the second division is found either in the sphenomaxillary fossa or as it courses around the back part of the maxilla; and by working backward the third or lower division may be found. But one must work out one's own details on the cadaver. Personally I find by introducing the needle immediately under the zygoma at the highest point of the curve and directing it toward the first finger of my other hand, which is placed on the opposite external angular process, in this way using the sense of position of the fingers to guide the needle and keep it at the proper angle, I have seldom had difficulty in finding the middle division of the nerve as it makes its exit from the foramen rotundum. For the inferior division the needle is introduced in the same spot but pointed to catch the nerve as it comes through the foramen ovale. As Patrick points out, the internal features of the human kind are variable, but in these cases one can be guided by recognizing with the needle the junction of the pterygoid process with the great wing of the sphenoid and working backward or forward until the nerve is engaged; in this way one gets a good idea of the depth to which it is necessary to go—as a general rule a depth of about 4 or 5 cm. is sufficient.

The needle should be withdrawn slowly with the stylet out, and if there is any deep oozing the stylet may be reinserted in the needle, and being pushed back to the place of oozing may be left there until the oozing stops. Any superficial bleeding may be controlled by pressure; the puncture wound may be closed by a drop of collodion.

Wilfred Harris, in England, practises injecting the ganglion itself through the foramen ovale with, he thinks, more permanent relief. His method is to push the needle through the cheek below the zygoma as low as possible through the bottom of the sigmoid notch in the lower jaw, the point being directed straight inward and upward until it hits the under surface of the sphenoid bone at the base of the skull. The point is then gently lowered by raising the handle and the edge of the foramen ovale is felt for at a depth of from 3.5 to 4.5 cm. according to the size of the individual. He asserts that in practically every case the needle can be made to pass through the foramen into the Gasserian ganglion if necessary. His reported results have been good.

THE SEVENTH OR FACIAL NERVE

Anatomical Relations.—The great motor nerve of the face has its nucleus in the lowest level of the pons, consisting of a long row of cells grouped together in the middle part of the *formatio reticularis*; its fibers stream dorsally and toward the medial line and then hooking around the nucleus of the sixth nerve in the floor of the fourth ventricle, run ventrally and laterally to make their exit close to the eighth nerve on the lateral aspect of the pons at its junction with the medulla. It runs with the eighth nerve in the internal auditory meatus, then passes in the Fallopian canal first outward to the inner wall of the tympanum then backward and downward behind the tympanum to the stylo-mastoid foramen. It may be separated from the tympanum by a very thin layer of bone, or this may even be incomplete, which explains the frequency of involvement of the facial nerve in disease of the middle ear, more especially as it gives off two small branches which enter the tympanum, one to the stapedius muscle and the other to the chorda tympani. Through the geniculate and otic ganglia connections are established with the fifth and the ninth nerves. The definite function of these connections is still obscure. Possibly, as Edinger suggests, the fibers conveying the sense of taste coming from the lingual branch of the fifth and the glossopharyngeal nerve reach in this way the small intermediate nerve of Wrisberg and the nucleus of the fasciculus solitarius.

The nerve supplies the occipitofrontalis, the muscles of the external ear, the stylohyoid and digastric, and divides to supply all the muscles of the face and the platysma.

The path connecting the cortex with the facial nucleus decussates in the upper part of the pons. It is probable that the bilaterally acting muscles of the upper part of the face have representation in both hemispheres, as they are seldom paralyzed in hemiplegia from a lesion of the pyramidal tracts above the pons.

Lesions of the facial nerve may be paralytic or irritative.

Facial Paralysis.—Etiology.—Paralysis of the seventh nerve—often called Bell's paralysis after the Englishman who first described it—frequently comes on after exposure to cold and is usually on that side of the face which has been more directly exposed to a direct draught of air. It is probably due to a congestion and swelling of the nerve in the narrow bony Fallopian canal causing compression of its fibers and paralysis. It appears, as a rule, in the course of two or three days after exposure, and is often associated with some pain behind the ear. Injury such as a blow on the side of the face or more frequently operations in the parotid region or for disease of the mastoid cells and the middle ear are often followed by facial paralysis. Its close proximity to the middle ear in some cases where the dividing lamina of bone is very thin, or even deficient, renders it almost impossible for the surgeon to avoid injuring it. As has already been said, disease of the middle ear itself may involve the nerve by extension.

Facial paralysis is occasionally associated with certain infectious diseases, as diphtheria, erysipelas, influenza, herpes zoster, and the recent epidemics of poliomyelitis have produced numerous cases caused by that disease. Syphilis is not an infrequent cause, but is usually associated with paralysis of other cranial nerves. It may be from involvement of the meninges, or a gumma involving the trunk of the nerve, and it sometimes appears as a manifestation of secondary syphilis, especially in women. The facial nerves on both sides may be involved in a general multiple neuritis.

Symptoms.—The muscles of the face on the side of the lesion not only lose their power, both in voluntary and emotional movements, but their tone as well; in other words, there is a condition of flaccid paralysis. The normal tonicity of the muscles of the opposite side causes an asymmetry of the face which is especially noticeable on any attempt to laugh or speak. In youths in whom the elasticity of the skin is still retained the asymmetry is not so noticeable when the features are at rest, but in elderly people it is very marked at all times. The nasolabial fold is flattened on the paralyzed side and the patient cannot raise that angle of the mouth. In attempting to show the teeth or to smile the mouth is drawn up to the healthy side. There is also disturbance in pronouncing all labials, and as the lips cannot be compressed properly, there is inability to blow out the cheeks, the air escaping from the paralyzed side; whistling is impossible. Owing to the involvement of the orbicularis palpebræ the patient cannot close the eye, and on attempting to do so the eyeball turns up (Fig. 40); during sleep, too, the eye remains partially open. The lower lid, also, especially in adults in whom the elasticity of the skin is lacking, falls away slightly from the eyeball so that the apertures of the tear duct no longer being in close apposition, the tears cannot be carried off and so they gather in the eye and overflow the cheek. Paralysis of the occipitofrontalis leaves the forehead smooth and an attempt to raise the eyebrows shows the inability to do so on the paralyzed side, the wrinkles of the forehead being marked on the healthy side and absent on the side of the lesion. The platysma is also affected in many cases, as may be shown by getting the patient to depress the lower lip forcibly. There is no deviation of the tongue when protruded, although there may be an apparent deviation, owing to the lips being drawn to the healthy side.

The electrical reactions are characteristic of a lesion of a peripheral

FIG. 40



Facial palsy.

nerve. In severe cases there is an absolute loss of faradic excitability of the paralyzed muscles in about ten days and the reaction to galvanism may be much diminished. In milder cases the reaction to faradism may simply be lowered and that to the galvanic current may be much slower than normal. It should be remembered that the faradic current stimulates only through the nerve terminations in the muscles, while the galvanic current stimulates the muscle fibers directly. Polar change is sometimes, though not always, seen, that is, the cathodic closing contraction becomes equal to or even less than the anodic.

Atrophy of the muscles follows as in nerve lesions elsewhere, but on the face it is scarcely perceptible, as the muscles normally are not very bulky.

If the lesion be in the pons there will probably be either involvement of the pyramidal tract and evidence of a spastic hemiplegia on the opposite side of the body or involvement of the sixth nucleus with consequent paralysis of the external rectus muscle and disturbances of the conjugate deviation of the eyes to the side of the lesion.

If it be on the surface of the pons there will be an associated nerve deafness. A lesion in the Fallopian canal between the geniculate ganglion and the chorda tympani branch would cause an associated loss of taste on the same side of the tongue in the anterior part. If none of these symptoms are present the lesion is probably in the lowest part of the canal or external to the skull.

One must differentiate now between a facial paralysis of peripheral origin, that is, from a lesion in the nucleus or nerve trunk, and one of central origin or a supranuclear paralysis. In the latter the lower part of the face is involved with asymmetry very similar to what one sees in the peripheral type, but while present for voluntary movement it may disappear in the movements expressive of the emotions especially if the lesion be above the level of the thalamus. There will be, especially in recent cases, a weakness of the orbicularis palpebræ, but there will be no weakness of the occipitofrontalis in raising the eyebrow. There will be no impairment of the reaction of the paretic muscles to electrical currents.

Course.—The onset of facial paralysis is, as a rule, fairly rapid, coming on in anywhere from a few hours to three or four days; it is frequently associated with a more or less severe pain behind the ear. It usually reaches its height in the course of twenty-four hours. The duration will depend on the severity of the lesion. In very mild cases a complete recovery may take place in two weeks or a month. In such a case the faradic excitability of the muscles is never lost. More severe cases may last several months and then, as a rule, there is not an absolutely complete recovery. The traces left may be but slight, but they will be perceptible on close examination. In many of these severe cases a certain amount of muscular contracture occurs as the muscles regain their voluntary power this does not occur when the paralysis remains complete.

This contracture is most marked in the muscles of the angle of the

mouth and by reproducing the nasolabial fold even to an exaggerated degree, produces the appearance of a paresis of the healthy side of the face. The appearance is strengthened by any slight action of the muscles being exaggerated, but immediately we ask the patient to forcibly raise the upper lip or close the eye, the weakness of the contracted muscles is very evident. This contracture of the facial muscles after paralysis is often associated with involuntary twitching of a spasmodic nature which may be very troublesome.

Treatment.—Our first endeavor in these cases, as in all peripheral nerve paralyses, must be to prevent stretching of the paralyzed muscles by the unopposed tonic action of their opponents, for two reasons: (1) First to retain as much as possible the elasticity of the paralyzed muscles, and (2) to prevent shortening of the healthy muscles, which may go on to a certain amount of fibrosis and contracture, producing a more or less permanent condition of deformity. In my own practice I have endeavored to meet this indication by the following simple contrivance and have found it very satisfactory from the point of view of the patient's comfort and in, I believe, shortening the course of the paralysis very considerably. The requirements are, two pieces of strong wire—ordinary strong hair-pins serve the purpose very well—and two pieces of adhesive plaster, or a piece of adhesive plaster and one piece of ordinary garter elastic. One hair-pin is straightened out and then formed to fit around the ear, the two ends overlapping in such a way that they can be bound and held in position by means of a piece of adhesive plaster. The second hair-pin is then bent about half an inch from its closed end so as to form a hook and the two limbs of the pin are then pressed together so that this hook will not be too bulky for the angle of the mouth. The two loose extremities are then bent toward each other at right angles. These two pieces can then be joined together by a piece of adhesive plaster doubled so that no adhesive surface is exposed, or better still by a piece of ordinary garter elastic about one-half or three-quarters of an inch broad and of the proper length to hold the angle of the mouth in position, suspended as it were from the ear, and thus overcome the asymmetry. The wire about the back of the ear can be padded with a little absorbent cotton and some may be inserted in the concavity of the hook as it lies in the angle of the mouth. This should be changed frequently during the day and the hook may be sterilized by dipping it into boiling water, being careful not to wet the adhesive plaster. This contrivance if properly fitted will be found to give a certain amount of comfort to the patients, and there is, as a rule, no difficulty in getting them to wear it all day, save at meal-time, and to sleep with it at night. The conscientious use of such a contrivance will certainly lessen the length of time of the paralysis, and I believe may prevent the occurrence of such cases as those recently reported by Toby Cohn, when in spite of the return of electrical reaction in the muscles, their functional power remained in abeyance, with persistent asymmetry and deformity of the face.

The unprotected condition of the eye sometimes results in slight

inflammation of the conjunctiva, produced by the entrance of irritants, but this usually subsides rapidly under treatment with a wash of acid. boric. and sod. bitor. The accumulation of tears in the eyes, caused by the eversion of the lower lid, probably acts as a protective, and it is not wise to interfere actively to prevent this. The patient should be instructed to close the eye with the hand when going to sleep.

One must of course at the same time endeavor to arrest or remove the cause of the disease. If there are other indications of syphilis, potassium iodide and antiluetic treatment should be energetically pushed. If due to tumor of the cerebellopontine angle this should receive surgical attention. If associated with middle-ear disease, free exit to the pus present should be secured; and if it follows exposure to cold the salicylates are indicated. Aspirin is useful in relieving the pain about the ear so often associated in the early stages of the paralysis and at the same time counterirritants in the shape of a mustard leaf or fly-blister, or, what I have found very satisfactory, a paint of equal parts of camphor and chloral hydrate.

Electrical treatment of the muscles should now be instituted, not that it has the slightest curative effect on the nerve lesion, but simply by the mechanical stimulation causing movement of the paralyzed muscles, their nutrition is maintained and atrophy prevented to some extent at least. If the muscles still react to faradism, that is, in a mild case, stimulation with this current may be used if the galvanic cannot be obtained, but it is better to use the latter in any case as it stimulates the muscles directly, and so, while it will stimulate the fibers through the nerve terminations just as well as the faradic, it will also stimulate those fibers whose nerve terminations have degenerated.

If the paralysis has been of some weeks' standing without any electrical treatment one should not give a bad prognosis simply because the muscles react poorly the first time of the treatment, as it will often be found that the reaction improves considerably after a few applications, and the functional power will often return after months of interruption.

The positive pole should be placed on any indifferent region: it may be held in the hand or placed on the back, and the negative pole used to stimulate the motor points of the various muscles (Fig. 45). It should be remembered that the mere application of the electrical current in itself is useless unless the muscles are made to contract by it.

If, as recovery takes place, contracture and overaction of the paralyzed muscles occur, the electrical treatment should be stopped as it will certainly not do any further good and will only increase the discomfort. It is very questionable, in my opinion, if a nerve-grafting operation is justifiable; the results so far of grafting the distal portion of the facial nerve into the spinal accessory, or the hypoglossal, have as yet not given sufficiently good results to be encouraging.

Prognosis.—In giving a prognosis, the nature of the cause, whether it is a progressive lesion and whether it can be removed, will of course influence one; but these points being decided favorably, one may state generally that if ten days or two weeks after the onset the muscles

still react to faradism the prognosis is good and complete functional recovery will occur inside of three months, the more exact time being judged by the extent of the reaction. If at that time the muscles do not react to faradism, recovery will probably not be absolutely complete and will be slower, the prognosis depending on how well they react to the constant current. In cases that have existed for some time without electrical treatment it will be well, before giving a prognosis, to test the reaction of the muscles more than once, as a muscle that has lain inert will not react at first very well but improves greatly in successive tests.

Facial Spasm.—It is absolutely necessary in the first place to differentiate between a facial tic and a spasm of the facial muscles, and this differentiation is not always easy owing to the similarity of their external form. Yet the establishment of a correct diagnosis is essential, since the prognosis and treatment are diametrically opposed. Tic is a psychical affection—a pathological habit, Brissaud termed it—and it is always associated with peculiar mental characteristics. It begins as a voluntary act and remains to some extent under control of the will. It remains a purposive movement, whereas a spasm has been defined as the motor reaction consequent on stimulation of some point in a reflex spinal or bulbospinal arc, the stimulus provocative of such spasm is itself of pathological origin. This spasm is confined to the distribution of the nerve, the oculomotor muscles and the tongue are not involved. It may be caused reflexly by painful affections of the cornea or conjunctiva, decayed teeth, or any affection of the fifth nerve—a tic might possibly develop from the same sources. Not infrequently it is due to a direct irritation of the facial nerve trunk, as from a tumor in the cerebellopontine angle or an aneurysm of the branch of the basilar artery. It has also been referred to as a late result of severe facial paralysis with only partial recovery. Facial spasm may also be caused by an irritating lesion of the cortical face centre and is then of the nature of Jacksonian epilepsy usually affecting the eyes and then spreading to the arm and leg centres producing the characteristic march of these attacks.

Symptoms.—Facial spasm may occur in various degrees of intensity the most simple is blepharospasm, which may commence as a rapid trembling or twitching of the lower eyelid which spreads gradually and in the course of time the upper lid is also affected until the whole orbicularis palpebrarum is involved, so that during the spasm the two lids approach one another, lessening the size of the palpebral fissure. Owing to the spasm the lacrymal duct is occluded and the tears may gather in the eye, but as soon as the spasm is over the eye is quite normal again. Such may be the first stage, but gradually other muscles in the supply of the nerve may also become affected, and coincidently with the blepharospasm we see an upward twitching of the angle of the mouth, a wrinkling of the side of the nose, and an elevation of the eyebrow on the affected side of the face. The spasms become progressively more frequent and more widespread, involving one bundle

of fibers after another until all the muscles supplied by the nerve may be involved in their totality. The face may remain free for some minutes or the twitchings may be practically constant. In early cases such spasm may last only a second, or in more severe cases it may last some minutes; often it may continue for hours. Although the contractions may be quite violent they are not associated with any pain. Voluntary effort has practically no influence in preventing or retarding the spasm, thus differentiating it from tic. Medication is usually without result; arsenic, the bromides, and gelsemium are of no avail; the effect of hyoscine is only temporary, and morphine is out of place.

Treatment.—In treatment, then, if no source of irritation can be found and removed, one may try galvanism, applying the negative pole to the back of the neck and the positive pole over the course of the nerve just in front of the lower part of the ear, and turning on the current gradually up to about 2 or 3 milliampères, maintain it for five or ten minutes and then gradually diminish again. This should be repeated daily. Unfortunately not much can be promised in most cases from the treatment, and in some cases it may be considered advisable to paralyze the facial nerve either by stretching it or preferably by the injection of alcohol. One then runs some risk of a permanent paralysis which the patient will not appreciate much more than his spasm. In many cases with the return of power comes a return of the spasm.

THE AUDITORY OR EIGHTH NERVE

The eighth cranial nerve is composed of two nerves which have quite distinct functions, we must therefore distinguish between the cochlear and the vestibular nerve, the former being the real auditory nerve and the latter subserving the function of giving us information concerning equilibrium and our relations in space.

Disturbances of Cochlear Nerve.—**Anatomical Relations.**—The cochlear nerve arises from the cells of the ganglion spirale. These cells send peripherally fine branches which arborize about the delicate hair cells of the organ of Corti, and centrally—analogously with the spinal sensory roots—their nerve fibers, having joined those fibers from the vestibule, enter the pons just laterally to the facial nerve in a single nerve root known as the *portio mollis* in comparison with the *portio dura* or seventh nerve. The fibers from the cochlear branch terminate in the tuberculum acusticum and in the ventral nucleus. From these nuclei the secondary paths cross to the other side in the trapezoid body as the *striæ acusticæ*, making connections with the superior olives and thence in the lateral fillet to the posterior corpus quadrigeminum and the external geniculate body. The former connection serves for the reflex arc with the various oculomotor nuclei, and the latter connects up with the cortical centres of hearing in the temporal lobes.

The vestibular nerve takes origin from ganglion cells in the labyrinth,

the peripheral branches of which terminate in the ampullæ and utricle of the semicircular canals, the central branches enter the pons with the cochlear nerve as the anterior root of the eighth nerve and run to the dorsal nucleus in the lateral part of the floor of the fourth ventricle, some fibers turn downward toward the spinal cord and others run directly to the anterior vermis of the cerebellum. From the dorsal acoustic nucleus connections are made through Dieters' nucleus with the cerebellar nuclei and also descending paths running in the anterolateral columns of the cord, which probably connect up with the anterior horn cells and function as reflex paths in the maintenance of equilibrium.

Etiology.—Theoretically disturbances of hearing may be caused by a lesion in any part of the course of the cochlear nerve or its end-stations in the brain, though, as a matter of fact, they are very much more frequently due to disease in the internal ear itself than to lesions affecting the second or third relays of neurones in the pons or cerebrum because of the bilaterality of representation necessitating usually more than one specially located lesion. A single lesion, a tumor growth for instance, might involve both posterior corpora quadrigemina and external geniculate bodies or might involve the pons sufficiently to cause a complete deafness. Foci of multiple sclerosis might also possibly produce the same effect, but when this occurs there is, as a rule, no difficulty in the diagnosis from the other signs of the disease present and there would be nothing to do in the way of treatment, at least as far as the deafness is concerned.

Of more practical interest are lesions affecting the nerve in its peripheral course. Thus meningitis may cause a nerve deafness, occasionally, though not always associated with a facial paralysis of the same side; the seventh nerve or portio dura is not as easily damaged as the eighth or portio mollis. Of more practical interest is its affection by tumor growth in the cerebellopontine angle which not infrequently arises from the sheath of the eighth nerve. In these cases one sees, of course, the general signs of intracranial tumor—headache, vomiting, and choked disk—and as localizing signs one finds complete nerve deafness with, as a rule, facial paralysis on the side of the lesion and not infrequently involvement of the fifth and sixth nerves on the same side. There is also, as a rule, complaint of vertigo of a characteristic and definite nature, and there may be increased deep reflexes in the opposite extremities with the abdominal reflexes diminished or lost, and in fact the usual evidences of an upper motor neurone paralysis on the side opposite the lesion—due to the pressure of the neoplasm on the pyramidal tract above the decussation of its fibers. The presence of a tumor in such a position should be easily recognized, and in the hands of a competent surgeon, with experience in cerebral surgery, the results of operation are good.

In the great majority of cases, however, where the nerve is affected, disturbances of hearing are due to disease of the internal ear, and this disease may be primarily in the internal ear or secondary to disease of the middle ear or inside the skull. Primary disease may be due to such

conditions as severe cold, a fall, or a blow, sunstroke or associated with malarial fever, typhoid, scarlatina, syphilis, measles, diphtheria, influenza, leukocythemia, Bright's disease, diabetes, and mumps. Secondary changes in the labyrinth may follow inflammation of the middle ear, or may be due to extension from intracranial disease as in cerebrospinal meningitis. Certain drugs such as quinine and salicylic acid taken in large doses or continuously, as well as the excessive use of tobacco may give rise to subjective noises or deafness. Deafness is often one of the symptoms of hysteria. Congenital deaf-mutism need only be mentioned here.

Treatment.—The consideration of the treatment of diseases of the middle ear and labyrinth belongs to the sphere of the otologist, and the reader is referred to special books on the subject. At the same time some points of general importance might here be mentioned. When the disease is in the external ear, that is, when it obstructs the passage of the vibrations of sound to the internal ear, but the latter is healthy, then the sound of the tuning-fork will still be perceived if it be placed on some bony part of the skull, but if it is held before the meatus its sound will be obscured. If, on the other hand, the labyrinth is diseased, then bone conduction will be relatively diminished or lost. Normally aërial conduction is better than bony conduction, that is, after the vibrations of a tuning-fork placed on the skull can no longer be perceived they will again be heard if it is held before the external meatus.

We have no means of distinguishing between disease of the labyrinth and a lesion of the nerve itself save by associated symptoms. In each case the deafness is the same and may be associated with symptoms of irritation—subjective sounds or tinnitus aurium. This symptom is often a most obstinate accompaniment of neurasthenic states and hysteria, probably because of the introspective and self-centred state of mind of these patients. It is also frequently a symptom of anemia, aneurysm of the cerebral arteries, or simply of a general arteriosclerosis, especially when, with a high maximum blood pressure, the minimum or diastolic pressure is relatively low. It is often caused by gout and syphilis. Individuals exposed to great noise, as machinists, boiler-makers, and so on, often suffer from tinnitus.

One's first efforts must be to remove or treat the disease of the labyrinth, and for this the reader is referred to books on otology. In gout, relief is sometimes obtained by alkalis and purgation; in syphilis, good results will follow energetic antiluetic treatment in early cases. In chlorosis and the anemias the proper treatment for these diseases will give corresponding relief to the ear symptoms, and in arteriosclerosis much can be done by levelling the blood pressure by appropriate means. Sometimes, in my experience, it is not essential to lower the maximum pressure to any extent, but relief is obtained if the minimum pressure rises to more normal relations with the maximum. Those suffering in consequence of habitual exposure to loud sounds should obtain quiet and rest or protect the ear by obstructing

the external meatus. In cases where no cause is discoverable, bromides, either alone or combined with belladonna and cannabis indica, have been recommended. Counterirritation over the mastoid in some seems to act beneficially. Section of the eighth nerve has even been attempted in the effort to cure the aggravating symptoms, but the results have in most cases been disastrous.

Disturbances of Vestibular Nerve.—Affections of this nerve are usually associated with disease of the cochlear, but, so far as one can separate them clinically, diseases of this nerve lead to disorders of coördination and vertigo, giving rise to a condition known as Ménière's disease. In this disease there is always more or less of an impairment of hearing with tinnitus and attacks of vertigo which come on with great suddenness, the individual falls as if struck by an unseen hand and may lie unconscious for a moment. The vertigo is of a very definite character, often the patient feels that he is turning around like a top, or in other cases surrounding objects seem to be rotating. It is often associated with nausea and vomiting and usually is followed by a severe headache. Frequently just before an attack there is an increase in the tinnitus, the roaring becoming higher and more shrill. These attacks vary in frequency, coming on in some cases daily, in others at much longer intervals.

Treatment.—The same treatment as has been suggested under disease of the labyrinth holds good for this condition, but the results are often far from satisfactory and operative treatment with removal of the semicircular canals is sometimes resorted to. Such radical measures of course result in complete deafness. Lumbar puncture has been recommended by Babinski as an effectual remedy, though it is difficult to see just how this would act. He recommends the withdrawal of from 3 to 20 c.c. of spinal fluid.

THE GLOSSOPHARYNGEAL OR NINTH NERVE

The glossopharyngeal nerve is a mixed nerve containing motor, sensory, and visceral fibers. The sensory fibers originate in the jugular and petrous ganglia and send one branch peripheralward and the other centrally to enter the medulla just laterally to the olive. They then penetrate in a medial and dorsal direction to end for the most part in the nucleus solitarius. Some fibers end in the dorsal nucleus in the floor of the fourth ventricle. The motor fibers originate, like those of the vagus nerve in the nucleus ambiguus, and after running dorsally and toward the median line turn laterally and then ventrally in a manner similar to what one sees in the seventh nerve and joining the sensory fibers pass out of the medulla on its lateral border. The nerve leaves the skull at the central part of the jugular foramen in a separate sheath of the dura mater external to and in front of the vagus and spinal accessory nerves. The superior or jugular ganglion is situated in the upper part of the groove in which the nerve is lodged

during its passage through the jugular foramen, and the inferior or petrous ganglion is situated in a depression in the lower border of the petrous portion of the temporal bone. It forms various connections with the vagus and the sympathetic nerves and also with the facial. It supplies the tympanum and the Eustachian tube with sensation through the tympanic (or Jacobson's) branch, and the pharynx possibly gets some of its sensory innervation also from its pharyngeal branch. It also conveys the fibers of taste to the circumvallate papillæ on the posterior third of the tongue; where these originate is still a matter of doubt. Its visceral fibers go to the carotid plexus and probably to the tonsils.

Our knowledge of the functions of this nerve is extremely limited owing to its close association and connection with the vagus and the fact that the nuclei of these two nerves and that of the eleventh nerve are all extensions of the same nuclear group. These nerves seldom suffer singly; the glossopharyngeal may be damaged by tumors at the base of the brain, or may be involved along with others in an inflammatory focus, a superior poliomyelitis. In abiotrophic conditions affecting the bulbar nuclei, *e. g.*, glosso-labio-pharyngeal paralysis, it degenerates with others.

THE VAGUS OR TENTH NERVE

Anatomical Relations.—The vagus is distributed to the great internal organs. It innervates the pharynx and esophagus, the larynx and the lungs, the heart, the stomach, and intestines. It contains not only motor and sensory fibers but visceral as well. For the histology and functions of this nerve we are much indebted to the recent work of L. R. Müller, in Augsburg. The motor fibers which supply the voluntary muscles of the pharynx and larynx undoubtedly originate in the nucleus ambiguus which lies in the medulla medialward and slightly dorsally from the substantia gelatinosa of Rolando and the descending root of the fifth nerve, the fibers first run dorsally and slightly medially and then hook around laterally and ventrally in the same way as the glossopharyngeal fibers do to become superficial on the lateral surface of the medulla just outside the olive.

The visceral fibers which innervate the visceral organs originate in the dorsal nucleus of the vagus in the floor of the fourth ventricle just lateral to the upper end of the hypoglossal nucleus; running ventrally and slightly laterally they become superficial with the motor root. They act on the various viscera through their association with cells of the sympathetic system type located both in the jugular ganglion and in the walls of the various organs. In this respect the visceral nucleus of this nerve represents the intermediolateral nucleus of the gray matter of the spinal cord, while the nucleus ambiguus represents the anterior horn cells. The sensory fibers have their trophic centres in the ganglion jugulare and the ganglion nodosum, thence the fibers

enter the medulla with the motor fibers and terminate about the nucleus of the fasciculus solitarius, which is the representative of the posterior horn of the cord.

It does not differ in this respect from other cranial and spinal nerves which contain fibers with vegetative functions distributed to the vessels, sweat glands and the erector pilæ muscles, the unstriped tissue of the iris and the ciliary muscles, and so on, but in the vagus these visceral fibers are derived from cells in its own ganglion, the jugular ganglion, and not from any sympathetic ganglion interposed, as is seen in the other nerves. The nerve makes its superficial origin in twelve to eighteen fine bundles of fibers from the side of the medulla behind the olive and below the glossopharyngeal fibers. These fine bundles unite to form one large root while still within the cranium, and there is formed a slight enlargement about the size of a pea, the jugular ganglion. It then passes through the jugular foramen to the outside of the cranium and gives off the meningeal and the auricular branches. Then there is a second enlargement, the ganglion nodosum. The vagus differs from the other cranial nerves, save the glossopharyngeal in having then two ganglia; possibly this may point to its being phylogenetically two nerves joined into one. Numerous connections with the ninth and eleventh and with the superior cervical sympathetic are formed here. It then takes its long course to the stomach, giving off branches to the lower half of the esophagus to form a plexus. The first branch from the vagus is the meningeal, which is given off immediately behind the ganglion jugulare and runs to the dura mater, which it probably supplies in part with sensibility and possibly it has vasomotor functions as well. The second branch is the auricular, running from the short part between the two ganglia of the nerve. It receives fibers from the glossopharyngeus and goes to the mastoid canaliculi and thence to the skull cavity. The auricular branch supplies the external auditory meatus with sensibility, and by irritation of the meatus coughing is caused by radiation of the irritation to the neighboring fibers of the superior laryngeal nerve.

After its exit from the skull cavity the vagus passes through the ganglion nodosum and gives off the pharyngeal branches. These form with the fibers from the glossopharyngeal nerve and with branches of the superior cervical sympathetic the plexus pharyngeus, which is chiefly motor in character and innervates the muscles (constrictores pharyngis, levator veli palatini, m. azygos, m. glossopalatinus, m. pharyngopalatinus). Fine fibers from the neighboring accessory nerve form connections with the ganglion nodosum and vagus stem and its branches to the pharynx.

The sensory innervation of the pharynx and larynx is almost completely supplied by the superior laryngeal nerve. This branch rises from the lower half of the ganglion nodosum. Often it gives off fine motor branches to the constrictor pharyngeus and sometimes branches to the cricothyroideus. In the throat it innervates the mucous membrane of the most posterior part of the tongue, the epiglottis and its

neighborhood, and that of the larynx. Through stimulation of these nerve endings, especially sensitive swallowing spots of the mucous membrane of the root of the tongue and the posterior wall of the pharynx, the swallowing act is started. This consists in a raising of the tongue against the roof of the mouth, and in a closing off of the nasal cavity by the palate; the epiglottis rises and with the posterior part of the tongue closes off the larynx. Thus both the sensory and motor part of the swallowing reflex arc are almost completely in the branches of the vagus. The centre for deglutition lies in the medulla, thence the fibers of the sensory vagus nucleus, the nucleus fasciculus solitarius, must connect with the motor nucleus (nucleus ambiguus) of this nerve. Reflex connection between the deglutition centre and the respiratory centre causes reflex inhibition of respiration during the act of swallowing. The feeling of air hunger is suppressed at the moment of swallowing, the latter taking precedence once the act is started.

The inferior or recurrent laryngeal nerve is the motor nerve to the larynx. It innervates all the laryngeal muscles except the cricothyroideus, also, it gives sensory branches to the trachea and numerous branches to the cardiac plexus and one to the aorta (nervus depressor). Although the fibers of the recurrent laryngeal branch are chiefly motor it contains also visceral and sensory ones, which will be considered more fully later. It may (according to Müller) convey fibers which innervate the vessels of the larynx or the thyroid gland. The tracheal nerves supply the mucous membrane of the respiratory passages with sensibility and set up the cough reflex when irritated. The function of the pulmonary plexus is least understood. The fibers of the vagus which go to the lung supply the bronchial mucous membrane with sensation—whether they act in regulating respiration—as Kernig and Brauer hold, is not demonstrated with certainty. Following bilateral section of the vagus, respiration becomes slower and deeper. Possibly this is because normally at the conclusion of inspiration expiration is excited through the vagus. Centripetal vagus fibers have an inhibitory action on the respiratory centre according to latest investigations. By stimulation of the pulmonary vagus inspiration is inhibited.

Action and Symptoms.—The centripetal fibers of the pulmonary vagus contain, as do the superior laryngeal and the tracheal branches, sensory fibers whose irritation (by poisonous gases) leads to inhibition of the respiratory centre. It is impossible to say whether these are sympathetic (viscerosensory) or purely sensory; probably they are sensory, as foreign bodies set up forcible coughing. The centripetal fibers of the pulmonary vagus do not serve for the conduction of the sensation of air hunger; section of the vagi does not give rise to cessation of respiration. The respiratory centre is responsible for the sensation of air hunger and it is excited not only by the diminution of oxygen in the blood but by the increase in the carbonic acid gas.

It is certain that the pulmonary vagus has centrifugal fibers and by stimulation of these fibers contraction of the unstriated bronchial muscle is produced. After section of the vagus the bronchioles become

dilated. We do not know what the exact physiological purpose of the bronchial musculature is. Its contraction under certain pathological conditions produces bronchial asthma and may lead under conditions of great emotional strain to a condition of dyspnea and stridulous breathing which when prolonged is termed asthma nervosum. By stimulation of the vagus an increase in the respiratory metabolism is produced and at the same time a slowing of the heart. The vagus has an inhibitory and slowing action on the heart. Section of both vagi causes increased frequency of the heart's action. Stimulation produces inhibition and cessation in diastole.

The cardiac inhibitory centre is in the medulla in the dorsal vagus nucleus and it can be excited (1) by increased intracranial pressure; (2) by psychical influences as the emotions—pleasure, for instance, slows and strengthens, worry hastens it; (3) by irritation of sensory nerves causing pain, whether spinal or viscerosensory; and (4) by sensory paths which run in the vagus branches to the heart, the so-called depressor cordis nerves.

As a matter of fact, stimulation of the central stump of the extra-cardiac vagus branches diminishes the frequency of the heart beat, and this disappears after section of the vagus. Later observations (Koster and Tschermak) show that the depressor breaks up into many branches on the aorta and is there influenced through increase in blood pressure producing a slowed heart action and lowered pressure. Viscerosensory fibers in the depressor cordis nerves form then a visceral reflex arc. The fibers from the sympathetic system to the heart act as antagonists of the vagus. Their stimulation increases the rapidity of the heart action. They come from the first dorsal especially and also from some of the higher ganglia; they run into the stellate ganglion and thence as non-myelinated fibers to the cardiac plexus.

I believe that the unpleasant sensations in the cardiac region, suffered by many patients with heart disease, are conveyed by these sympathetic fibers to the cord and thence to consciousness. The pain in the chest and down the inside of the left arm in the area of the eighth cervical and the localized tenderness so often present in this region are explained by irradiation of the excitations which come to the upper dorsal and lower cervical cord through these sympathetic fibers. The vagus does not carry unpleasant sensations from the heart.

The vagus supplies not only the pharynx but the lower and middle parts of the esophagus. There is no unanimity of opinion as to whether its action results in the opening or closing of the esophagus and the cardia. Some time after section of the vagus the loss of tone of the cardia and the disturbances of swallowing are recovered from. But we must understand that the musculature of the esophagus and the cardia carries its own innervation organs, and the fibers of the sympathetic and the vagus have only an exciting or inhibitory action on these ganglion cells.

Action on Stomach.—On the stomach musculature the vagus has a stimulating action. Section of the vagus in the neck of a dog slows

the movements in the stomach wall (Pawlow). After some time, however, the movements return to normal. Artificial stimulation of the vagus increases gastric peristalsis. Irritation of sensory nerves anywhere in the body, when associated with pain, produces inhibition of the stomach movements; emotional excitement may act similarly. We should remember that the vagus does not innervate the stomach musculature directly, but, as in the case of the esophagus, acts on the ganglion cells lying in the stomach wall. The vagus may also start up antiperistaltic movements and the act of vomiting. Vomiting doubtless depends on the action of a definite centre in the medulla. By increased intracranial pressure or other diffuse disturbance in the brain, *e. g.*, concussion, we see "cerebral vomiting" due to the irritation of the medullary centre. Emotional disturbances with the sensation of nausea may also excite this antiperistaltic centre of the medulla.

Action on Gastric Secretion.—The influence of the vagus on the gastric glandular secretion is very important. It is common knowledge that the sight or smell of food starts up secretion of the gastric juice. Pawlow has shown that in dogs the sight of meat or bread sets up in a short time a richer secretion of gastric juice than does the sight of milk. This must take place through the vagus, because, after section of the vagus, secretion from the peptic glands ceases, but commences again on stimulating the peripheral end of the cut vagus. There is no evidence to show that the vagus nerve contains centripetal fibers from the stomach to the brain. The tenderness and pain in the hypochondrium and to the left of the spine, so common in gastric disease, is no doubt due to the impulses conveyed to the cord by the sympathetic radiating into the corresponding nerve-root distribution. The vagus has an exciting influence on the peristalsis of the small intestine in opposition to the splanchnic nerves which inhibit its movements.

Action on Pancreas.—Its action on the pancreas is not certain but according to Pawlow stimulation of this nerve causes an increase in the secretions of this gland. The sympathetic fibers also cause an increase of the secretions of the pancreas, but there are differences in the secretion from that produced when the vagus is stimulated. With the latter the juice is more concentrated and contains more ferments. Fibers from the vagus go to the liver, chiefly to the gall-bladder and Glisson's capsule. Their function is not known with certainty, but if a needle be introduced experimentally into the vagus nucleus in the floor of the fourth ventricle, sugar is excreted in the urine after a few hours. This would suggest that the vagus was responsible, but on the other hand it occurs if both the vagi are cut, and does not occur if the splanchnic nerves have been severed.

Action on Kidney.—Little is known of the influence of the vagus on the activity of the kidneys. Arthaud and Butte have found it to have an inhibitory action on the urinary secretion, and section of the vagus below where the cardiac branches are given off, gives rise to an increase in the secretion, probably through the uncontrolled vasodilator action

of the sympathetic branches arising from the dorsal segments of the cord. In this connection it is interesting to note the general antagonism between the vagus and the general sympathetic system. Irritation of the vagus causes contraction of the bronchial musculature, while that of the sympathetic gives rise to widening of the lumen of the bronchi. The vagus inhibits the heart's action, the sympathetic through its *nervi accelerantes* produces a quickening. The splanchnic sympathetic inhibits the small intestinal peristalsis and the vagus excites it.

The vagus centres may, as has already been shown, be influenced by increased intracranial pressure causing a slowness of the pulse, heightened blood pressure, and often cerebral vomiting. It may be influenced by emotional conditions, such as extreme pleasure, pain or nausea. It would appear also to be responsible largely for many of the symptoms associated with shock.

The centre also may be involved in new growths or vascular lesions, such as thrombosis of the posterior cerebellar artery, or in inflammatory lesions such as one finds in poliomyelitis, but it is seldom affected alone in these conditions. Superficially the nerve fibers may be involved in a meningitis of any nature, often giving rise to an irregularity of the pulse.

In the various neuroses it is most commonly affected, producing the well-recognized symptoms of cardiac, gastric, or intestinal neurosis, either through its own functional disturbance or through its failing to control the activity of the antagonistic sympathetic system.

Treatment.—The treatment of diseases affecting the vagus will be the treatment of the underlying condition, whether it be of an organic nature, or a mental and emotional condition. Therapeutically, atropine has a paralyzing action on the peripheral terminations of the vagus fibers, while adrenalin exerts a stimulating action on the sympathetic fibers.

THE SPINAL ACCESSORY OR ELEVENTH NERVE

The spinal accessory nerve arises from two sources—the medullary portion is in reality a part of the vagus arising from the same nuclei, while the spinal portion originates in a long column of cells lying in the lateral part of the anterior horn in the upper five cervical segments of the cord. These bundles of fibers ascend to the brain to join the root of the medullary portion and make their exit with it from the cranium through the jugular foramen. It supplies the sternocleidomastoid muscle and part of the trapezius. It may be involved in conditions at the base of the brain or in caries of the upper cervical vertebræ with the other nerves, but the more common sources of its paralysis are in the neck, glandular enlargements, and exposure to cold.

The symptom of paralysis of the spinal part of this nerve is the paralysis of the two muscles it supplies. The sternocleidomastoid

turns the head to the opposite side and tilts the chin, and the trapezius moves the head backward and toward the same side and elevates the shoulder. The accessory nerve innervates only the clavicular and acromial portion of this muscle, as a rule, but this is apparently variable.

The diagnosis is made by the evidence of weakness in these muscles with the consequent atrophy and changes in their electrical excitability.

Treatment.—The treatment will depend on the cause of the paralysis; when this is only temporary, electrical treatment to the paralyzed muscles with the galvanic current is indicated. If contracture has occurred with the production of wry-neck or torticollis, hot applications followed by massage and passive movements will in some cases tend to relieve it. If, on the other hand, the condition is of too long standing, tenotomy of the contracted muscle and forcible correction of the deformity may have to be resorted to. Spasmodic torticollis, more properly called tic, must be differentiated from this condition, as it does not represent a diseased condition of these nerves, but is dependent on a mental condition entirely, and will be taken up in another chapter.

THE HYPOGLOSSAL OR TWELFTH NERVE

The hypoglossal nerve is the motor nerve to the tongue, it arises from its nucleus, extending from the lower part of the fourth ventricle to the closed-in portion of the medulla, where it lies just anterior to the central canal. Its fibers run ventrally and emerge from the medulla in numerous small bundles in the groove between the olive and the pyramids. It leaves the skull by the anterior condyloid foramen and runs some distance beneath the internal carotid artery and internal jugular vein on its way to supply the various muscles of the tongue. It forms connections with the vagus and the upper three cervical nerves.

Paralysis of the functions of the hypoglossal nerve may be either supranuclear, nuclear, or infranuclear. Supranuclear paralysis results from a lesion of the opposite cortex in the inferior Rolandic region or of the corticonuclear tract in the internal capsule, the crus, and the upper part of the pyramidal tract. It is usually associated with a paralytic weakness of the extremities on the same side as that of the tongue, as in lesions of the upper motor neurones; elsewhere there is no resulting atrophy. The tongue on being protruded deviates to the paralyzed side owing to the action of the healthy geniohyoglossus muscle.

Nuclear disease is usually of a degenerative nature and is generally bilateral (Fig. 41). The nucleus may, however, be involved in an inflammatory focus as in polioencephalitis or by new growths. However, as the two nuclei are so close together, they are usually both involved, causing an atrophic paralysis of the whole tongue; protrusion is impos-

sible, articulation is interfered with, and swallowing more or less difficult. The nerve itself may be involved in the medulla by vascular lesions or tumor growths, or at the base of the brain by meningeal neoplasms; or in its course in the neck by tumors or stab wounds, or in caries of the first and second cervical vertebræ. In infranuclear lesions there results an atrophic paralysis of the homolateral side of the tongue. When lying in the mouth the paralyzed side may lie higher than the healthy side at the root, owing to the paralysis of the hypoglossus. The paralyzed side is smaller and has a pitted hummocky appearance and on palpation feels soft and flabby. If the lesion has affected the

FIG. 41



Atrophy of the tongue due to bilateral lesion of the hypoglossal nucleus.

nerve fibers in the medulla there will often be a hemiplegia of the opposite side of the body owing to the coincident destruction of the pyramidal tract. If the nerve be affected after it leaves the medulla the atrophic paralysis of one side of the tongue is usually associated with paralysis of the palate and vocal cords of the same side.

Treatment.—The treatment will depend on the cause of the paralysis. When this can be removed, as in luetic conditions, it will be advisable to keep the tongue muscles in as good a state of nutrition as is possible by electrical stimulation.

LOCALIZATION OF ORGANIC DISEASES OF THE SPINAL CORD

In the study of diseases of the nervous system, more perhaps than in any other branch of internal medicine, a careful systematic examination of the patient is necessary. Many of the symptoms are evident to very casual observation, and the temptation to make a snap diagnosis

frequently leads to serious mistakes. The history of the case should be taken, a general examination of the patient made, and the condition of the nervous system investigated. In taking the history, and during the general examination, much will be learned about the patient's mental condition, his intelligence, disturbances of cerebration, the presence of insane ideas, hallucinations, etc., and any defects of speech, dysarthrias or aphasias will be noted, and further investigations may later be made along such lines as may be indicated. The functions of the cranial nerves should then be investigated systematically and in order; and following this the motor system, the sensory system, and the reflexes, and finally the gait and any trophic disturbance should be noted, and inquiries should be made respecting bladder and rectal symptoms. Electrical examination is sometimes of diagnostic value.

From the *extent and nature of the symptoms* we form an opinion as to the *seat of the disease*, i. e., we localize the lesion or form an *anatomical diagnosis*. From the *history of the mode of onset* and other features of the case we diagnosticate the *nature of the lesion*, i. e., we make a *pathological diagnosis*. We are thus in a position to form a broad conception as to the disease in the case we are investigating.

Anatomical Diagnosis.—PERIPHERAL NERVES.—Let us begin with the peripheral part of the nervous system—the peripheral nerves—and let us consider a mixed nerve, say the median. We know it is made up by the union of the outer and the inner cords of the brachial plexus and thus gets fibers from several spinal roots from the fifth cervical to the first dorsal. We know it supplies all the superficial muscles on the front of the forearm except the flexor ulnaris and some small muscles of the hand (flexor profundus digitorum, flexor sublimis, flexor longus pollicis, pronator quadratus, abductor, opponens and outer head of the flexor brevis pollicis, and the first and second lumbricales). We know also that it supplies the skin over the distal parts of the first two fingers, the palmar surface of the thumb, and a part of the radial side of the palm of the hand.

Head and his fellow-workers have shown also that the sensory fibers in the peripheral nerves can be divided into three systems.

1. "*Deep*" Sensibility.—Those which subserve "deep" sensibility, and conduct impulses produced by pressure and the movements of the parts. The fibers of this system run mainly with the motor nerves and are not destroyed by division of all the sensory nerves to the skin.

2. "*Protopathic*" Sensibility.—Those which subserve "protopathic" sensibility and respond to painful cutaneous stimuli and to extremes of heat and cold, but they *do not* enable us to form any definite appreciation of the locality of the spot stimulated. (Similar visceral protopathic fibers pass to the internal organs.) Protopathic fibers from adjacent nerves overlap each other to a considerable extent.

3. "*Epicritic*" Sensibility.—Those which subserve "epicritic" sensibility, fibers which endow the skin with sensibility to light touch. They conduct the impulses which enable us to localize the position of cutaneous stimuli, to discriminate two points of a compass and to

appreciate minor degrees of heat and cold. There is little overlapping in the distribution of these fibers.

When the sensory impulse reaches the spinal cord it becomes shunted into tracts devoted to the conduction of impulses grouped in a way different from that found in the peripheral nerves. It is no longer a question of protopathic, epicritic, or deep sensibility. The tracts in the central nervous system are devoted to the conduction of impulses concerned with pain, heat, cold, and touch, and the sense of positions of the muscles. The fibers for these various forms of sensibility travel up more or less well recognized paths in the cord. Entering by the posterior roots the sensations of *pain*, *heat*, and *cold* travel up in the gray matter a distance which varies with each form and then cross over in the posterior commissure to the opposite side and continue to ascend in the anterolateral tract. *Tactile* sensibility evidently has more than one pathway up the cord, both crossed and uncrossed, while the *sense of position* of the muscles is conveyed by the posterior columns and travels up to the nuclei of these columns on the same side on which it enters the cord. Certain fibers in the posterior roots form connections directly or through the mediation of a second neurone with the anterior horn cells of the same segment, subserving the *reflex arc* and the *maintenance of tone* in the muscles.

Motor Neurones.—With regard to the motor apparatus we have in the lateral columns the *crossed pyramidal tract* and a smaller column in the anterior part of the cord, the *direct pyramidal tract*, conveying the upper motor neurones from the Rolandic cortex. It was formerly supposed that these connected up directly with the motor cells of the anterior horn, but Schäfer has shown that this is not the case, but that they terminate about the cells of Clarke's column and the cells of the medial portion of the gray matter. Probably through the mediation of these cells they form their connections with the cells of the anterior horns.

These *upper motor neurones* convey voluntary impulses from the motor cortex to the anterior horn cells, and also have a certain restraining, inhibitive action on them, so that a lesion of the upper motor neurones will of course prevent the transmission of voluntary impulses and thus cause paralysis, but will not prevent reflex action; in fact, the reflexes will be accentuated and exaggerated owing to the withdrawal of the cerebral restraint, and the tone of the muscles will also be increased. Atrophy of the muscles does not occur, and the electrical reactions are not altered.

The motor cells of the anterior horns of the gray matter are the trophic centres for the muscles; lesions of these cells cause paralysis with atrophy of the muscles they supply, with changes in their electrical reactions, and of course with the consequent break in the reflex arc; from the falling out of the motor part there is loss of tone and absent reflexes.

The nutrition of the bones, joints, and skin is affected by lesions of the posterior nerve roots (as is seen in tabes), but irritation of the root

has more effect than simple loss of function, and a lesion in front of the ganglion that is nearer the cord is more liable to cause trophic changes than one behind it. Thus in acute inflammatory lesions of the cord, intense changes of nutrition are likely to affect the skin below the level of the lesion—bed-sores and sloughs form rapidly and with very slight provocation.

Reflexes.—The reflexes are dependent on the integrity of the reflex arc. It has been shown that in the knee-jerk and other tendon-jerks the response is too rapid to allow the impulse to travel through this arc and it is said therefore that they are dependent on the tone of the muscles, but the tone is itself dependent on the integrity of the reflex arc, therefore the tendon-jerks may also be said to be dependent on this integrity. It is certain that a break in the arc always causes a loss of the tendon-jerk. The reflex arc for the triceps and wrist-jerk is at the level of the sixth and seventh cervical segments; that for the knee-jerk at the second, third, and fourth lumbar segments; the ankle-jerk at the level of the fifth lumbar and first sacral; the bladder reflex is in the third sacral and the anal reflex in the fourth sacral segment.

The so-called superficial reflexes, the abdominal, epigastric, and plantar, are cerebrospinal reflexes and not simply spinal, their reflex arc is made up of the sensory nerve to the cord and thence by some afferent path to the brain, returning by the pyramidal tract to the anterior horn and thence to the muscle. We expect to find these absent below the level of the lesion in cases of transverse lesions of the cord involving the pyramidal tracts, and also in cases of hemiplegia, especially when the paralysis is fairly complete, these reflexes are diminished or absent on the paralyzed side; in fact in any cases where the pyramidal tracts are fairly completely destroyed these reflexes will be impaired.

Localization in Transverse Section of Cord.—If now there is a lesion in the centre of the cord, such as occurs in syringomyelia, involving the posterior commissure and encroaching as it so often does on the gray matter of the anterior horns in places, one can see how it will cause a loss of sensibility to pain, heat, and cold over varying skin levels, as the paths for these sensations from the same skin area pass in the commissure at different levels. Tactile sensibility is not necessarily affected as there are alternate paths for its transmission up the cord. If the anterior horns of the gray matter are involved one will get a corresponding atrophic paralysis of the muscles supplied. This is commonly seen in the back muscles and produces curvature. It may affect any other part as well. If the pyramidal tracts are involved by this or any other lesion we get a spastic paralysis below the site of the lesion, with increased reflexes and the extensor plantar response (Babinski's phenomenon).

In amyotrophic lateral sclerosis (*d*, negative; *μῦς*, a muscle; *τροφή*, nutrition); as the name shows, we have evidence of a lesion of the anterior horn cells and also of a degeneration with sclerosis of the pyramidal tracts, but no sensory loss.

In acute poliomyelitis ($\pi\acute{o}\lambda\acute{o}\varsigma$ = gray + $\mu\epsilon\lambda\acute{o}\varsigma$ = marrow) we have evidence of a lesion of the anterior horns with consequent atrophic paralysis—loss of tone, loss of reflexes and electrical changes, and no sensory loss.

In subacute combined sclerosis—such as we occasionally see associated with severe secondary or septic anemias where, speaking generally, there is degeneration in the posterior columns and the pyramidal tracts chiefly, one gets both ataxia and spasticity—an ataxic paraplegia; this is not the name of a disease but simply of a symptom.

With a lesion causing a hemisection of the cord one gets loss of sensibility to pain, heat, and cold on the opposite side below the lesion, and a motor paralysis with increased tone and reflexes and Babinski's plantar phenomenon on the side of the lesion, with loss of sense of position on this side as well.

Localization in Vertical Direction.—So much for localization in the transverse direction in the cord. We must now consider the question of localization in the vertical direction. Now one must remember that although it is not so evident to superficial observation, still the human body is built on a segmental plan similar to what we see in the earthworm. The spinal cord illustrates this especially well and it is necessary to put out of one's mind the distribution of the peripheral nerves, made up as most of them are by contributions from several segments, and to think in segments. The roots, both anterior and posterior, *i. e.*, motor and sensory, are given off from the cord on both sides and the origin of each of these represents a segment of the cord, and it has been shown by clinicopathological observation and experimentation that each segment has definite functions, each supplies a definite area of skin with sensibility, each contains motor cells for certain muscles. The muscles, especially of the extremities, being intersegmental, *i. e.*, functioning to cause the movement of one segment with another, receive, as we should expect they would, innervation from several segments of the cord, and the accompanying tables show more or less accurately the muscular representation in the cord.

Each segment of the cord supplies a definite area of skin. These areas overlap somewhat, but by means of cutting two alternate posterior roots of the same side, the full distribution of the segment between these two has been definitely studied—careful clinical observation and postmortem study of cases of transverse lesions of the cord have also of course added materially to our knowledge on this subject, and we must add also the work of Head and Campbell in observing the distribution of the eruption in herpes zoster and later finding the characteristic lesion in the corresponding posterior ganglion, have also been of considerable help. From these studies we have comparatively definite knowledge of the segmental sensory localization in the cord. The following charts compiled by H. H. Tooth from the records of cases in the National Hospital for the Paralyzed and Epileptic, London, England, are in my experience accurate (Figs. 42, 43, 44, and 45).

CHART I

Segments.	Muscles.	Segments.	Muscles.
First cervical	Rectus capitis lateralis Rectus anticus major Rectus anticus minor Obliquus superior Obliquus inferior Sternothyroid Sternohyoid Omohyoid Splenius Complexus	Sixth cervical	Longus colli, scaleni. Scaleni. Pectoralis. Subscapularis. Teres major. Latissimus dorsi. Deltoides. Brachialis anticus. Biceps. Coracobrachialis. Serratus magnus. Extensor carpi radialis longior. Triceps. Anconeus. Supinator longus.
	Obliquus inferior Sternothyroid Sternohyoid Omohyoid Splenius Complexus Sternomastoid		Longus colli. Scaleni. Serratus magnus. Triceps. Anconeus. Supinator longus. Extensor carpi radialis longior. Pronator quadratus. Flexor longus pollicis.
Second cervical	Sternothyroid Sternohyoid Omohyoid Splenius Complexus Sternomastoid	Seventh cervical	All deep muscles in front of forearm except inner half of flexor profundus and all superficial muscles in front of forearm except flexor carpi ulnaris, viz., flexor radialis teres; flexor carpi radialis, palmaris longus, flexor sublimis digitorum.
	Sternothyroid Sternohyoid Omohyoid Splenius Complexus Levator anguli scapulæ Platysma Sternomastoid Diaphragm		All muscles of radial and posterior brachial regions, viz., extensor ossis metacarpi pollicis.
Third cervical	Sternomastoid Diaphragm Scalenus medius	Eighth cervical	Extensor brevis pollicis, extensor indicis. Extensor longus pollicis, extensor communis digitorum. Extensor minimi digiti.
	Rhomboideus Subclavius Supraspinatus Infraspinatus Longus colli Scaleni Pectoralis Subscapularis Teres major Latissimus dorsi Deltoides Brachialis anticus Biceps Coracobrachialis Serratus magnus		Pronator quadratus. Flexor longus pollicis. Deep muscles in front of forearm except flexor profundus, and all superficial in front of forearm except flexor carpi ulnaris as in seventh.
Fourth cervical		First dorsal	All muscles of radial and post-brachial region as in seventh segment, also flexor carpi ulnaris.
			Flexor profundus. Abductor, opponens, and flexor brevis pollicis. Flexor brevis minimi digiti. Abductor minimi digiti. Lumbricales.
Fifth cervical			

CHART II

Segments.	Muscles.	Segments.	Muscles.
First dorsal	Adductor, opponens, and flexor brevis pollicis Adductor minimi digiti Lumbricales	First sacral	Peroneus tertius. Extensor hallucis proprius. Soleus. Popliteus. Adductor minimi digiti. Flexor brevis digitorum. Abductor hallucis. Interossei and lumbricales.
First lumbar	Abdominal muscles (lower part) Quadratus lumborum Psoas		Extensor longus digitorum. Peroneus tertius. Extensor hallucis proprius. Soleus. Popliteus. Abductor minimi digiti. Flexor brevis digitorum. Abductor hallucis. Interossei and lumbricales. Ischiocavernosa.
Second lumbar	Cremaster Psoas	Second sacral	Ischiocavernosa. Accelerator urinæ. Sphincter vesicæ. Sphincter ani.
Third lumbar	Psoas Pectineus Sartorius Iliacus Gracilis Quadriceps extensor Adductor longus and brevis	Third sacral	Accelerator urinæ. Sphincter ani. Sphincter vesicæ. Levator ani.
Fourth lumbar	Pectineus Sartorius Quadriceps Adductor magnus Obturator externus Tensor vaginæ femoris Semimembranosus Tibialis anticus	Fourth and fifth sacral	
Fifth lumbar	Semimembranosus Tibialis anticus Gluteus medius and minimus Pyriformis Obturator internus Gemelli Gluteus maximus Semitendinosus Gastrocnemius Tibialis posticus Flexor longus digitorum		
First sacral	Pyriformis Obturator internus Gemelli Gluteus maximus Semitendinosus Gastrocnemius Tibialis posticus Flexor longus digitorum Biceps Peroneus longus and brevis Extensor longus digitorum		

Segmental Distribution.—It will be seen that the segmental distribution about the trunk is very evident, but on the limbs it does not appear so at first sight. If one, however, remembers the embryonic

FIG. 42

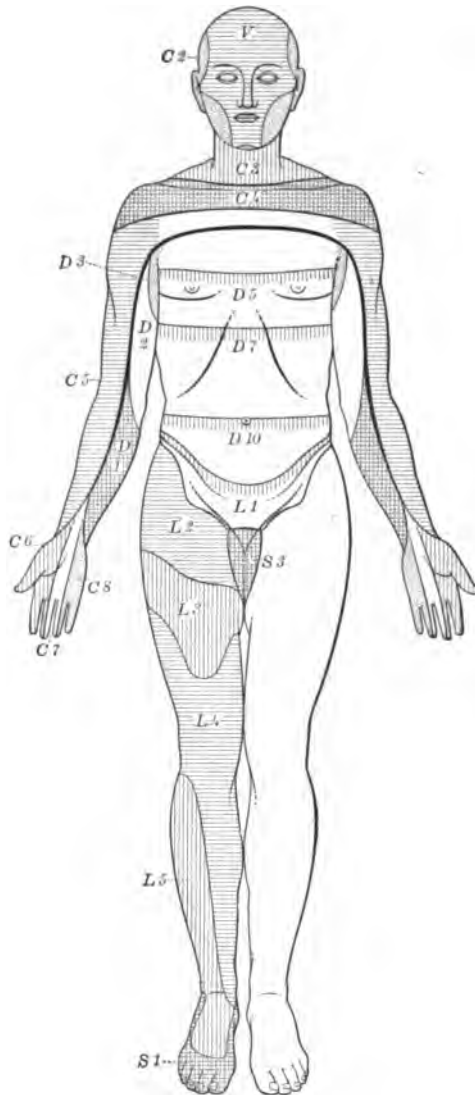


Chart illustrating the segmental spinal sensory areas. Front view.

position, that we are originally four-footed creatures, with the extremities at right angles to the body, and rotated so that in the upper extremities the radial border points forward, and with a similar rotation

in the lower extremities, one can see immediately how the segmentation is followed out. The distribution of the first four cervical segments is simple, then with the arm in the embryonic position the

FIG. 43

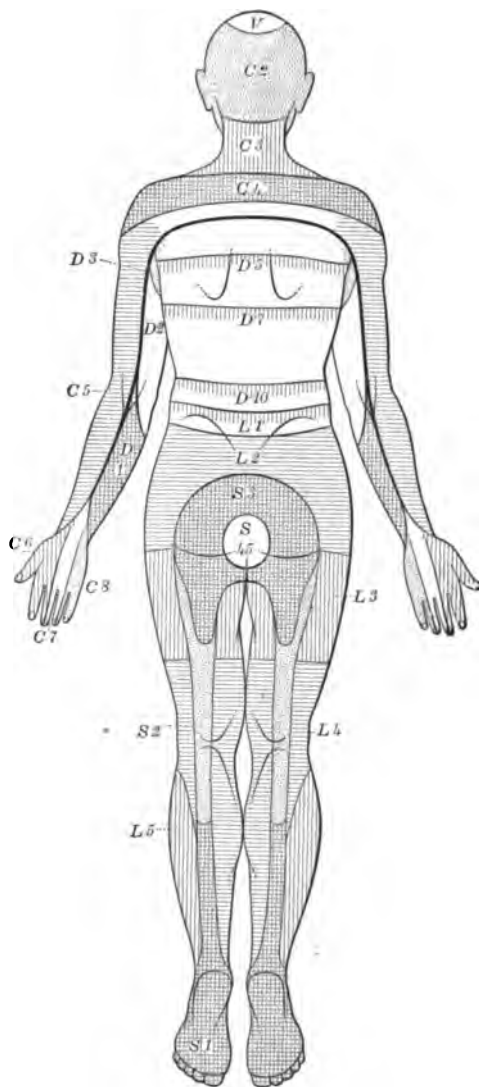


Chart illustrating the segmental spinal sensory areas. Rear view.

fifth cervical is distributed over the anterior part, the sixth slightly caudalward and so on with the rest of the cervical segments and the three first dorsal, all of which are distributed in their proper order to

the extremity. In this way the fourth cervical, and the fourth dorsal areas become, practically speaking, adjacent on the trunk at about

FIG. 44

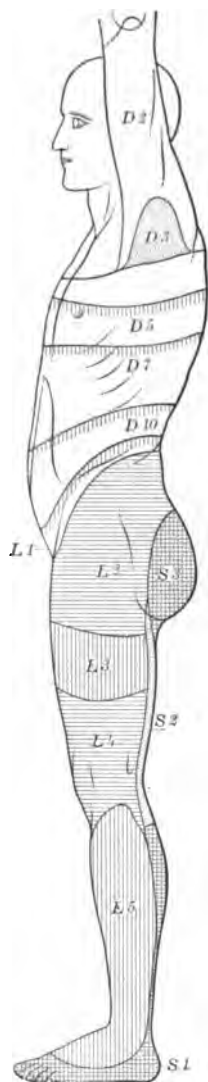


Chart illustrating the segmental spinal sensory areas. Side view.

FIG. 45

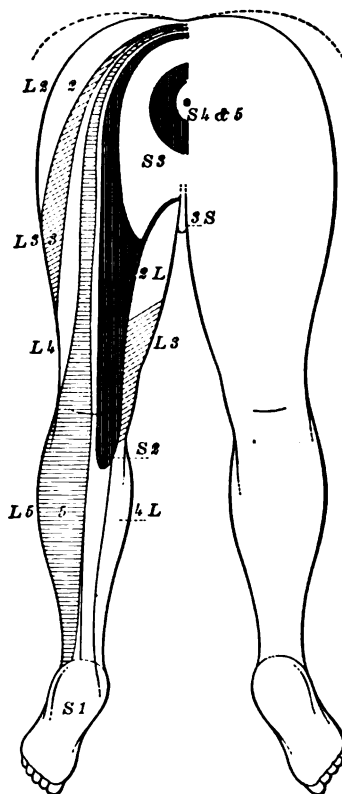


Chart illustrating the segmental spinal sensory areas of the lower extremities.

the level of the second costal cartilage. The same thing holds good for the lower limbs, when in the embryonic position the anus is at the

lower extremity of the body, and the distribution of the last two sacral segments are immediately about this point. The third sacral segment surrounds this and is distributed over the buttocks in a saddle-shaped area, and so on, as is shown in Fig. 45, as we proceed cephalward.

Each segmental area is distributed in its proper position if we conceive the body to be in the embryonic position. Thinking of it in this way there is no difficulty in carrying in the mind a fairly accurate picture of the segmental sensory distribution. It will only be necessary to remember such landmarks as the second costal cartilage where the fourth cervical and the fourth dorsal areas come together—and the ensiform cartilage is approximately at the level of distribution of the seventh dorsal segment, the umbilicus is at the tenth dorsal level, the fifth lumbar segment is distributed over the outer side of the calf of the leg.

Localization of Level of Lesion.—In localizing the level of a lesion in the cord it should be remembered that each segment overlaps to some extent, especially as far as tactile sensibility is concerned, so that if the lesion of the cord be fairly complete, it will, by stopping the passage upward of sensory stimuli, cause more or less complete loss of sensibility below the level of the lesion. Yet there will be a narrow area at the upper margin where sensibility is simply impaired, and it is the upper border of this that will indicate exactly the highest level of the lesion in the cord. Thus a lesion at the level of the first dorsal segment would cause a loss of sensibility in the inner half of the arm and forearm to the wrist, and on the body below the second costal cartilage level. As the ocular fibers of the sympathetic leaves the cord at this level, there would also be seen alteration in the size of the pupil. In the case of a paralyzing lesion the pupil would be small, and the palpebral fissure would appear narrow owing to the retraction of the eye caused by paralysis of Müller's muscle. There would also be noted an atrophic paralysis of the small muscles of the hand, especially of the thumb. If the lesion be absolutely complete all reflexes are abolished below the level—this is the case also in sudden irritating lesions, even when incomplete, for a time at least—otherwise there will be a spastic paralysis below the level of the lesion, with increased tendon-jerks, absence of the abdominal and epigastric reflexes, and the presence of an extensor plantar response (Babinski's phenomenon). The bladder will act reflexly and without voluntary control, and there will be retention of urine with the incontinence of overflow.

Relation of Spinous Processes to Segments.—For the purpose of treatment when surgical interference is indicated, it is necessary to remember that though the cord in the embryo extended down to the end of the spinal canal, it did not continue to grow in length to such an extent as the body did, so that in the adult its various segments no longer lie opposite their corresponding vertebræ. The following table of Sir Victor Horsley's will give the localization of the segments in relation to the spinous processes of the vertebræ:

	Superficial origin of spinal nerve roots.	Apices of spinous processes of vertebrae.	
Cervical,	1		Cervical.
	2	1	
	3	2	
	4	3	
	5	4	
	6	5	
	7	6	
	8		
Dorsal,	1	7	Dorsal.
	2	1	
	3		
	4	2	
	5	3	
	6	4	
	7	5	
	8		
	9	6	
	10	7	
	11	8	
	12	9	
Lumbar,	1	10	
	2		
	3	11	
	4		
	5		
Sacral,	1		
	2	12	
	3		
	4		

Except in very stout people it is usually easy to count the spinous processes, but it is sometimes well to remember that the third dorsal spine is on the level of the commencement of the spine of the scapula, and the seventh is on a level with the lower angle of the scapula. A line drawn transversely across from the highest levels of the crests of the iliac bones will pass through either the fourth lumbar spine or the space between the third and fourth spines.

Pathological Diagnosis.—So much for the localization of the lesion—the anatomical diagnosis. Let us now consider the pathological diagnosis—the nature of the disease. For this we are dependent to a large extent on the history of the onset of the condition. It is obvious that a tumor will not give pressure symptoms in the course of an hour or so with no previous history of symptoms nor will a hemorrhage take weeks to become complete. Vascular lesions come on suddenly—inflammatory ones are acute or rarely subacute. In compression of the cord from tumor growths, cysts, or vertebral caries, the history of the onset is usually subacute or chronic. In the degenerative conditions—the abiotrophies and systemic degenerations—months or years elapse before the symptoms reach their full development.

The etiology and history are usually of great assistance in making the diagnosis. Hemorrhage (hematomyelia) usually follows injury to the spine, though in some rare cases it may take place into a syringomyelic tumor without any previous history of injury, but it is sudden. Thrombosis is, as a rule, not quite so sudden in onset and often other evidences of syphilis are present or a history can be obtained. Injury

of course may cause a fracture and dislocation of the vertebræ with compression of the cord and hemorrhages into its substance or from the meningeal vessels. A previous history of syphilitic infection is important, as some diseases of the cord, such as *tabes dorsalis*, and some forms of meningitis and softening, are dependent on this etiology. The presence of tuberculosis elsewhere in the body is often of some importance, as vertebral caries or a tuberculoma of the cord often are secondary to a focus elsewhere. Acute myelitis is not infrequently associated with some infection, as gonorrhea, the exanthems, influenza, and tuberculosis. Subacute combined sclerosis is often associated with severe secondary anemia, both probably being caused by the same toxin. In the abiotrophies a family history of a similar condition, often through several generations, is frequently obtainable, as in Friedreich's ataxia, family spastic paraplegia, etc.

The age of the patient is often of some importance. Disseminated sclerosis usually begins before forty; Friedreich's ataxia usually before twenty, and the majority of cases of acute poliomyelitis are three years old or under, though it often occurs under the age of fourteen and sometimes affects adults. *Tabes*, save in rare cases of inherited lues, has seldom occurred before twenty.

Before leaving the consideration of the spinal cord it will be interesting to study the innervation of the various viscera. It has already been mentioned that they possess a certain degree of sensibility, similar to what Head has called protopathic sensibility in the peripheral nerves, *i. e.*, pain can be recognized and extreme degrees of heat and cold, but there is no definite localization of the stimulus possible. The viscera receive this sensibility through cells lying in the intermedio-lateral tract of the gray matter of the cord, the axis-cylinders of which pass out in the anterior roots and pass in the fine communicating branches to the sympathetic ganglia, where they terminate by arborizing about the sympathetic ganglion cells. Some fibers from these return to the spinal nerves and run to the periphery, others go to the various viscera. The nerves to the heart, for instance, are derived from the first three dorsal segments, and when these nerves are irritated, the irritation is conducted to the posterior roots of the dorsal spinal nerves with which they are connected, and gives rise to referred pain in the region of distribution of these segments over the inner side of the arm, so commonly complained of in *angina pectoris*. In visceral disease pain and tenderness occur in the skin area supplied by afferent root cells of the same spinal segment in which the visceral afferent root cells are situated. The antagonistic action of these sympathetic nerves to the vagus has been more fully discussed under the latter nerve.

ACUTE POLIOMYELITIS (ACUTE ANTERIOR POLIOMYELITIS, INFANTILE SPINAL PARALYSIS)

Acute poliomyelitis is an infectious disease caused by a specific organism, characterized by an atrophic paralysis of skeletal muscles

to a greater or lesser extent, and this paralysis may come on more or less abruptly. It reaches its maximum in a few hours, as a rule, and then tends toward recovery in some parts and the formation of permanent contractures and disability in others.

The relation of the disease to a lesion of the spinal cord was first recognized in 1840 by Heine, but the fact that apparently only sporadic cases occurred, and a fatal issue was very rare, rendered the pathogenesis of the disease a mystery until within the last few years, when the disease began to appear in a most severe epidemic form. Possibly epidemics had occurred before but had escaped recognition. Norway and Sweden from the years 1903 to 1906 were smitten with a series of epidemics which have been most thoroughly observed and described by Wickman and Harbetz and Scheel. In these epidemics over a thousand cases were observed with a mortality of 13.1 per cent. Within the last five years severe epidemics have occurred in New York, in 1907, consisting of about 2500 cases, and again in 1908, when about 1200 cases were observed. In Victoria, Australia, in the autumn months of 1907, Dr. Stevens observed 135 recent cases. In Massachusetts during the summer and fall of 1908, Lovett and Emerson collected 234 cases, and in Salem, Virginia, Wiley and Dardem reported 25 cases in an epidemic which occurred the same summer. Vienna and lower Austria suffered from a similar epidemic in 1908, when Zappert found data from 266 cases available. Epidemics of moderate severity are reported as having occurred in 1909 in Marburg and also in Westphalia and in France. The following article is based on personal experience gained in the observation of a series of 45 recent cases of the disease in an epidemic that affected Montreal and its environs in the summer and fall of 1909.

Symptoms.—Males are quite as frequently affected as females, but age is an important factor—though perhaps not as important as formerly supposed. In the series referred to, 60 per cent. of the cases affected were three years of age or under; at the same time 10 per cent. of the cases were in adults. Certainly this disease should no longer be called infantile paralysis. Nationality certainly gives no immunity; epidemics have been reported in practically every known country in the world. Poverty with its associated overcrowding and more or less heat and dirt seems to be a contributing factor in the spread of the disease, possibly through the numerous flies and mosquitoes attracted by such places. In any case the majority of cases seem to come from such surroundings, and the fact that most of the cases appear in August and September, practically no cases occurring during the winter months, would point to the same conclusion.

Bacteriology.—Great advances have been made in the last two years in the study of the etiology of the disease. First, Landsteiner and Popper, and later Flexner and his fellow-workers in the Rockefeller Institute, have been able to inoculate monkeys with the disease. The latter workers in a most brilliant series of experiments have succeeded in transmitting the disease through a series of monkeys from one to another; they have shown that the etiological factor is not a soluble

toxin but an ultramicroscopic virus similar to the organism of rabies. They have been able to cultivate this organism and have caused the disease in monkeys by inoculating this culture. They have shown that this virus will penetrate a Berkefeld filter, and have demonstrated that it retains its virulence after having been frozen for a considerable time. They have yet to show us how it gains an entrance into the system. In the attempt to learn this they have shown that cultures made from the mucous membranes of the nasal passages in monkeys suffering with the disease will cause the disease when inoculated into other monkeys; but cultures from other tissues, for example, lymph nodes, will do so too. They have also shown that if the mucous membranes of the nasal passages be scarified and smeared with infected tissue, the monkey may get the disease. But this is not sufficient proof that the disease is air-born, and one is immediately struck with the objection that if the disease be air-born why does it die out in the winter months, as the virus has been shown not to be influenced by freezing for a considerable time. One must be impressed with the idea that the disease must be carried by an intermediate host that is active in the summer and autumn months and inactive during the winter season, and probably of world-wide distribution. The house fly, or some of the common species of flies, would answer well to all these attributes. When we consider also that all the other diseases that are known to be caused by a filtrable virus, as for instance dengue, Mexican typhus, yellow fever and rabies, are all carried by an intermediate host and gain admittance into the system of the individual by inoculation from a bite of one of these, our impression should be greatly strengthened that in acute poliomyelitis the same process takes place. Since this article went to press it has been shown experimentally by Prof. J. N. Rosenau that the stable fly *Stomoxys calcitrans* may be a carrier of the disease.

Pathology.—Macroscopic Anatomy.—In recent cases the brain when removed from the cranial cavity is edematous and looks much too large to fit back into its place, and there may be some congestion of the superficial vessels. The cord in a recent case, *i. e.*, one that has died during the acute onset, if palpated even before opening the dura, will be felt to be quite hard at the affected parts, usually the cervical or lumbar enlargements, often both. There may be some congestion of the vessels of the pia-arachnoid, too, at these levels. I have observed also a transverse wrinkled appearance of these membranes as if a fine thread had been wound tightly and irregularly around the cord, evidently due to the extreme edema present. On section through the affected part there will be more or less eversion of the edges of the cord, and there will be evident to the naked eye a softening of its tissues with congestion of the vessels, so that the gray matter is no longer distinctly demarcated. Such lesions may affect the whole length of the cord or may be more or less localized to one or two parts. Similar lesions may affect the medulla, pons, or the various parts of the cerebral or cerebellar hemispheres. The spleen is usually enlarged and its

lymphoid tissue is strikingly prominent. The kidneys may show simply a cloudy swelling or as in one of my cases an acute hemorrhagic nephritis.

Microscopic Anatomy.—The leptomeninges, it may be only of the cord but often of both brain and cord, show a marked round-celled infiltration, chiefly in the neighborhood of the anterior spinal artery and penetrating into the anterior fissure along with the vessel, but it is present also on the posterior aspect of the cord. The spinal vessels are markedly congested and there is an excessive perivascular lymphocytic exudation. There is no evidence of thrombosis in any of the vessels. In the substance of the cord the vessels are dilated and congested generally, not only in the region of the gray matter but in the lateral and posterior columns as well. They are in every case surrounded by numerous round cells, mostly lymphocytes; and there are also a great many granular cells among these. A few ganglion cells still exist with a fairly normal outline. But apart from these there may be seen cells in various stages of destruction, some large and swollen, staining poorly with the nucleus almost extruded, others small and shrunken, staining deeply with no nucleus visible, others again scarcely visible on account of the numerous small round cells that encompass them, evidently the final stage in their disappearance. The congestion of the vessels and perivascular exudation of lymphocytes is more evident in the anterior horns on account of the better vascular supply, but the individual vessels in the posterior columns show an exactly similar condition. Small hemorrhagic extravasations are not infrequent. The ground substance of the gray matter has a rarified appearance with dilated meshes due to the edema present. (Plate XLI.)

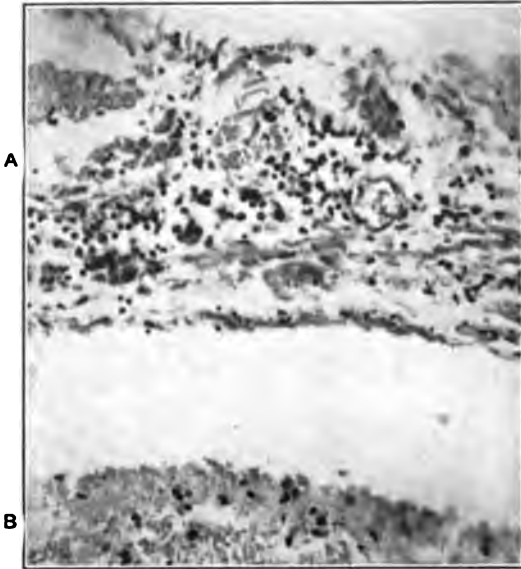
Such lesions may be found either localized to one part or, as is the rule in early fatal cases, more or less general in the cord and medulla and not infrequently in the pons and brain.

If the examination be made some few days after the subsidence of the acute symptoms very much the same appearance will be present, but the lymphocytic infiltration will not be so evident, especially in the white matter of the cord. The granular cells will be much less numerous, and the clumps of phagocytes, having demolished those ganglion cells that were too severely damaged, will have disappeared, and the evidences of inflammation will have subsided.

Onset.—In the majority of cases the disease was preceded by certain prodromal symptoms which lasted on an average a little over two days before the onset of the paralysis. In some cases it is true the paralysis developed suddenly while the child was playing around apparently in its usual health, and in these cases the first thing noticed was that the child fell down. This accident, usually of quite an insignificant nature, was naturally looked upon by the parents as the etiological factor, but in view of our knowledge of the disease, and the insignificance of the accident, it must be considered as *post hoc* rather than *propter hoc*, as the result rather than the cause of the paralysis. In other cases the prodromata were present for six or twelve hours before

PLATE XLI

Fig. 1



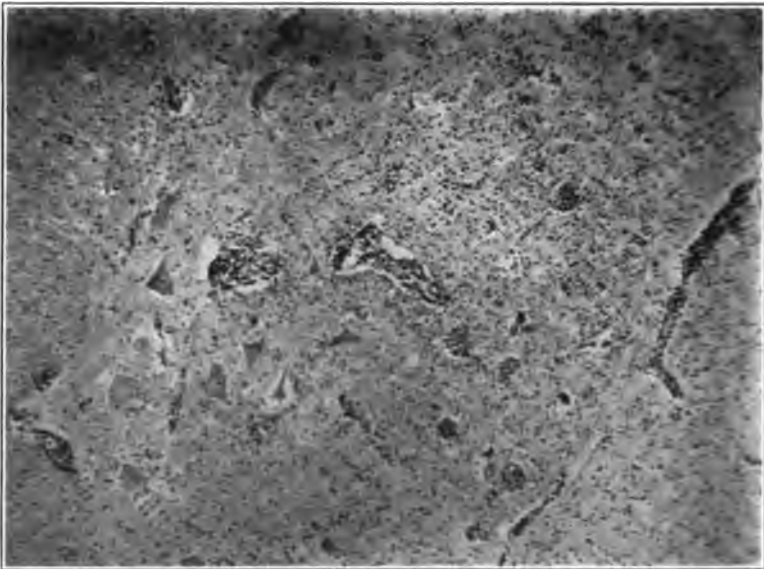
Acute Poliomyelitis, showing: A, Associated Meningitis; B, Edge of Cord.

Fig. 2



Acute Poliomyelitis, showing the Vascular Congestion and the Surrounding Infiltration. The Anterior Spinal Artery.

Fig. 3



Acute Poliomyelitis. The Various Stages of Destruction of the Anterior Horn Cells.

the onset of the paralysis; in others, again, it was as long as five to seven days after the onset of the prodromal symptoms before the paralysis of the limbs appeared.

As a general rule the patient complains of a little indefinite general malaise, has some headache, is restless, and may vomit once or twice. The bowels are usually constipated, but in a few cases there has been diarrhea. Some indefinite gastro-intestinal disturbance so common in children is suggested, especially as the fever is only slight as a rule; but if the child be old enough to make a complaint, or if in younger ones the limbs be moved, there is immediately evidence of pain and tenderness in one or other or perhaps all the extremities, which should suggest something more than the ordinary gastro-intestinal disturbance of infancy. Delirium may follow upon the restlessness; more commonly apathy supervenes. In about 30 per cent. of the cases there has been a history of more or less marked clinical signs of meningitis; in only 3 cases were there convulsions.

According to Flexner and his fellow-workers, in this stage a blood count will show some leukocytosis with a relative increase in the lymphocytes, and examination of the spinal fluid gives a positive Noguchi butyric acid test showing the presence of globulin. Cytological examination of the cerebrospinal fluid shows an increase of white cells, both polymorphs and lymphocytes, which is of great diagnostic value especially in the abortive cases to be described later.

As a rule the paralysis at its onset, or within a few hours of its onset, is at its height, but in some cases there is a spread of the disease quite similar to what one sees in the so-called Landry's paralysis, spreading up from the legs to the trunk, involving the upper extremities and finally the respiratory centres of the diaphragm. In others again there seems to be an extension laterally of the original lesion in the cord or mid-brain, which may superimpose a spastic paralysis of the legs on a flaccid paralysis of the arms or trunk muscles, by involvement of the pyramidal tracts. Or, if the original lesion be in the midbrain causing cranial nerve paralysis on one side of the face, there may arise in addition and from the same cause a hemiplegia of the opposite side of the body. In one of our cases this hemiplegia appeared only eight days after the onset of the cranial nerve paralysis. Such a delayed extension of the disease is, however, rare.

Types.—As Wickman pointed out, there may be several types of the disease depending on the relative severity of the infection and on the part of the nervous system affected.

1. **Spinal Type.**—There is first the spinal type of the disease, by far the most common, in which the lesion is confined to the cord, causing paralysis of variable extent and distribution. One or all or any combination of the extremities may be paralyzed, or in the more fortunate cases even a few muscles in one extremity may be picked out in an isolated manner. It is in these cases that not infrequently we see a progression of the disease simulating Landry's paralysis. The lesion first affecting the lumbar enlargement, as a rule, and spreading up the

dorsal region causing paralysis of the muscles of the back and the abdomen, the intercostals, the arms and shoulder girdle, the neck, and finally the diaphragm, the so-called Landry's type of the disease. (Plate XLII.)

2. **Meningeal Type.**—A meningeal type has been recognized in which the symptoms of cerebrospinal meningitis mask the prodromal period of the disease, this later develops into the spinal type.

3. **Pontine and Medullary Type.**—The pontine and medullary type of the disease is less common; it may be pure or in some cases is combined with the spinal type or the cerebral type. In these cases we have any combination of cranial nerve paralysis. While affection of the facial nerve is the most common result of this type, oculomotor paralysis is not uncommon, and the motor branches of the fifth nerve are occasionally affected, causing an inability to close the jaws, as in one of our cases.

4. **Cerebral or Encephalitic Type.**—The cerebral or encephalitic type is in my experience still more uncommon. The onset may be more or less sudden, as in the other types of the disease with fever, and the development of stupor or coma, and frequently convulsions. The fontanelles bulge; the neck is stiff and the head retracted; strabismus may develop; in some cases also we may see hemiplegia, depending on the site of the inflammation.

5. **Cerebellar Type.**—A cerebellar type has also been described in which there results the characteristic weakness of the homolateral extremities with loss of tone and ataxia.

6. **Neuritic Type.**—In some cases the associated pain is so severe and later the tenderness so excessive and the atrophy so rapid that there appears to be a condition of neuritis associated with the lesion in the cord. With the onset of the paralysis the cytological and chemical examination of the cerebrospinal fluid changes, as Flexner has shown, both the globulin content and the number of white cells diminishing rapidly to the normal.

7. **Abortive Cases.**—Wickman described *abortive cases* of the disease as early as 1905 in the epidemic in Norway and Sweden, which he has worked up so thoroughly; but there has until recently been some doubt as to the authenticity of these cases which have the prodromal symptoms similar to the other cases but develop no paralysis. But in the past year Flexner has shown as a result of his experimental work that a diagnosis can be arrived at by a chemical and cytological examination of the spinal fluid during the acute stage. And he has shown more positively still in later stages that the cerebrospinal fluid of cases of poliomyelitis, whether active or abortive, has a neutralizing action on the virus, so that when the infective virus is mixed with the cerebrospinal fluid from a recovered case, and allowed to stand for a little, it loses its infectivity and no longer causes the characteristic symptoms and paralysis when inoculated into a monkey. Drs. Anderson and Frost, of the Public Health and Marine Hospital Service and others, have put this into practice with very satisfactory results. Six out of 9 of

PLATE XLII

Fig. 1



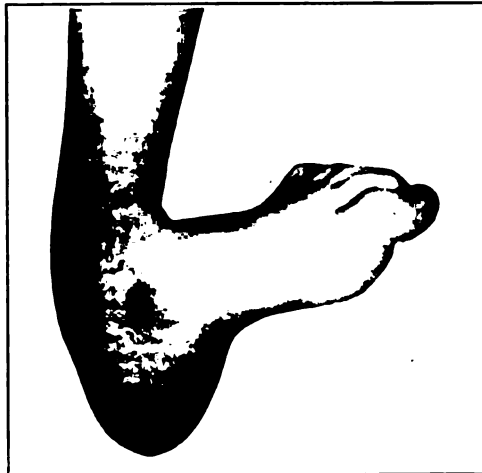
Acute Poliomyelitis, showing Paralysis of the Abdominal Muscles in a Child.

Fig. 2



Acute Poliomyelitis. The Subsequent Atrophy of the Affected Muscle

Fig. 3



Acute Poliomyelitis. Contracture of the Anterior Tibial Group Following Paralysis of the Calf Muscles.

their cases who had recently recovered from suspected poliomyelitis without paralysis (abortive cases) showed the same germicidal action as the serum from a frank case of poliomyelitis.

Following this acute stage the fever subsides and the child seems better, but there is more or less widespread flaccid paralysis and great tenderness if the affected parts be manipulated. This, as a rule, passes away fairly rapidly save in the neuritic type. The paralysis, too, tends to improve somewhat more slowly, and in the great majority of cases only partially, so that in two or three weeks while there has

FIG. 46

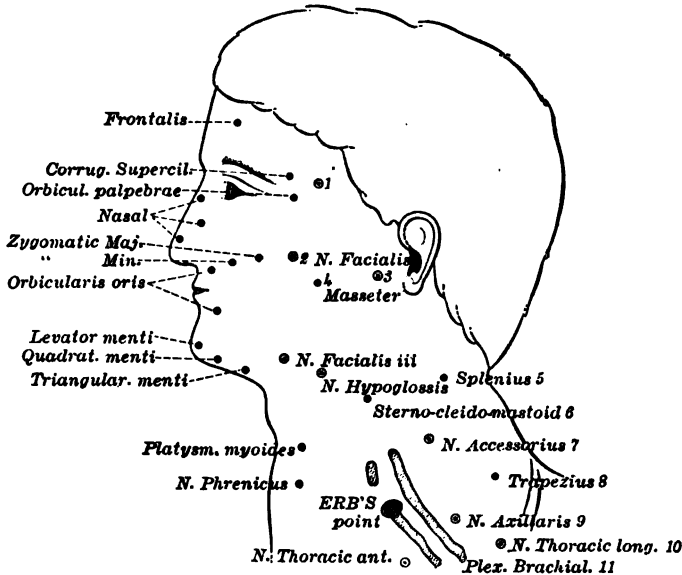


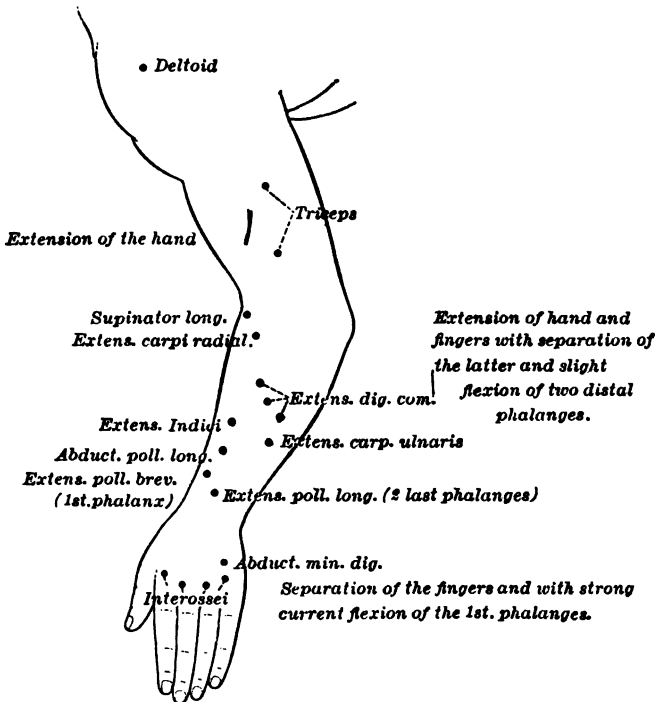
Chart showing the motor points for stimulating the muscles by the galvanic current: 1, contraction of the muscles of the forehead and the eyelids; 2, contraction of the muscles of the nose and upper lip; 3, contraction of the muscles of the whole half of the face; 4, closure of the jaws; 5, turning of the head to the opposite side; 6, turns face to the opposite side, the ear approaches the shoulder of the same side; 7, flexes head to the side, raises the shoulder, protrudes the lower jaw; 8, raises the shoulder and draws the scapula toward the spine; 9, contraction of the deltoid; 10, serratus action, scapula pushed forward and out; 11, contraction of almost all of the arm muscles. Erb's point: Get action on the biceps, deltoid, brachialis ant., and supinator long.

been great improvement in the extent of the paralysis as compared with that present after the acute stage a disabling amount still remains. When the respiratory muscles are paralyzed an intercurrent pneumonia very often causes a fatal issue at this stage. While the power in some muscles continues to improve, in others wasting appears. (Plate XLII.) The tendon-jerks are absent in the affected parts. There is usually no demonstrable impairment of sensibility to tactile or painful stimulation. The changes in the electrical reactions of the paralyzed muscles are characteristic. If they still react to the faradic

current two weeks after the onset of paralysis they will regain their power, but frequently they have lost the reaction to the faradic and show characteristic changes in their reaction to the galvanic current, the reaction of degeneration.

Contractures are now apt to appear unless proper and efficient treatment be instituted. The unparalyzed or partially paralyzed muscles, when their opponents are more completely paralyzed so that their opposition is lost, tend to shorten and may give rise to fearful deformities. (Plate XLII, Fig. 3.)

FIG. 47



Motor points for the muscles of the dorsal surface of the arm and hand.

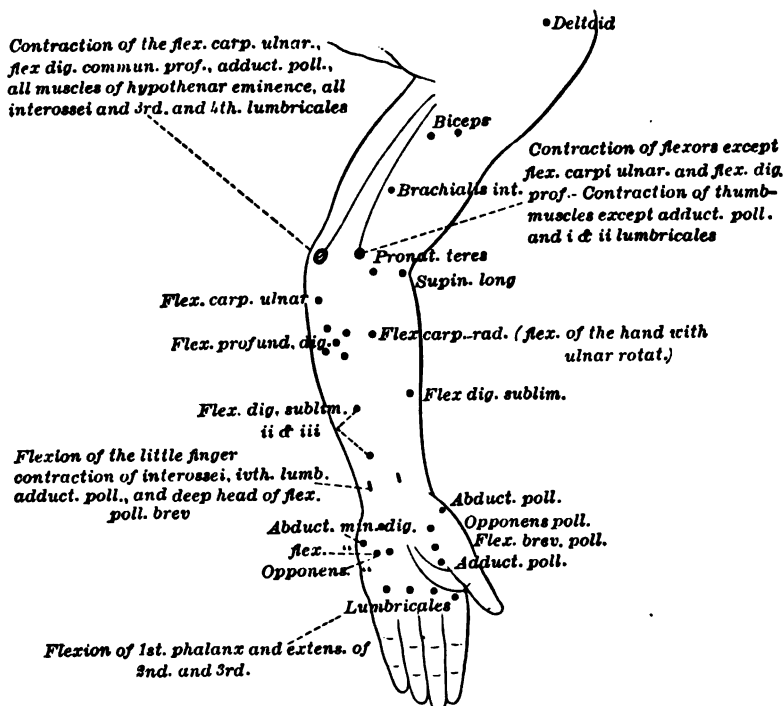
A second attack of the disease is unknown and Flexner has shown that patients who recover from an attack have developed an active immunity.

Diagnosis.—The great difficulties in diagnosis are in those abortive cases referred to, which undoubtedly must be the chief sources of the spread of infection, because, as a rule, unrecognized. A differential blood count showing a relative increase in the lymphocytes should be very suggestive, especially in the presence of an epidemic of the active disease, and of course the examination of the cerebrospinal fluid would

be more convincing. After the termination of the illness, the neutralizing action of the blood serum on the filtrate of the active virus, so that it would no longer cause the disease in monkeys, would of course be conclusive evidence.

Rickets in infants may give some trouble in differentiation, but the appearance of the child, the reflexes, and especially the electrical reaction of the muscles should solve the problem. The same holds good for rheumatic affections and osteomyelitis. Peripheral neuritis has a more gradual onset and is, as a rule, symmetrical.

FIG. 48

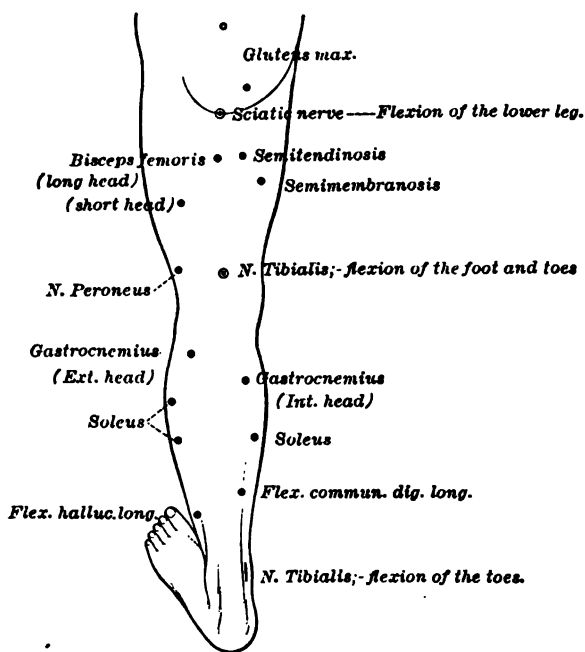


Motor points for the muscles of the palmar surface of the arm and hand.

Treatment should be divided into three parts, first, *prophylactic*; and in view of the arguments advanced under the section on bacteriology it would be advisable until we know more about the method of entrance of the virus into the system to take means to prevent the exposure of infants and children to the bites of flies and mosquitoes. This can be accomplished by the proper regulation of garbage disposal and the removal of filth; also by the proper protection of houses in an afflicted district with fly screens. The use of mosquito netting over the beds at night should also be advocated. At the same time disinfection of the nasal passages with a 1 per cent. solution of hydrogen

peroxide would not do any harm and would be on the safe side. Flexner has also shown that urotropin (which, as Cushing showed, is broken up and excreted into the spinal fluid as formalin) when given by mouth some time before the monkey was inoculated with the active virus, seemed to lengthen the incubation time of the disease very considerably and in some cases to prevent the onset of paralysis entirely. Whether or not we will ever have an immunizing serum against the disease is still in the laps of the gods.

FIG. 49

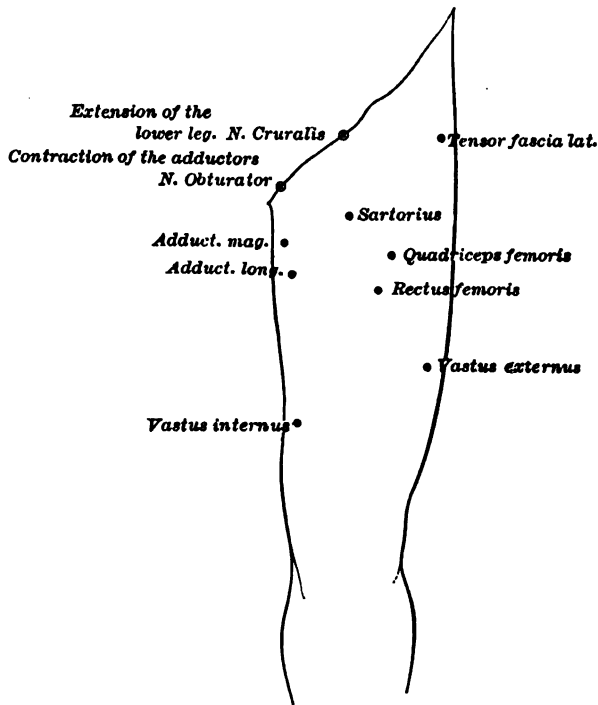


Motor points for the muscles of the posterior surface of the leg.

Treatment during Stage of Onset.—During the stage of onset we are practically powerless. Urotropin should be pushed in the hope that by its disinfecting action on the cerebrospinal fluid it may limit in some degree the activity of the virus. Lumbar puncture, theoretically, might be of some use, especially after the onset of the lesion in the cord and the appearance of paralysis, in reducing, possibly, the edema of the cord which in itself must lead to a certain amount of destruction. It is very questionable whether the lateral position or the prone position of the patient makes one particle of difference to the condition going on in the cord, and the same may be said for ice-bags along the spine and counterirritation, which only serve to add pain and discomfort to misery. The bowels should be opened with a brisk mercurial purge, and the diet should be light and easily digested. Very often

when the signs of meningeal irritation are marked, lumbar puncture will give relief to the headache, and hot baths will relieve the pain and rigidity of the muscles, giving a chance for a restful sleep. In other cases pain may be relieved by the exhibition of salicylates or phenacetin or in some cases an opiate may be necessary.

FIG. 50

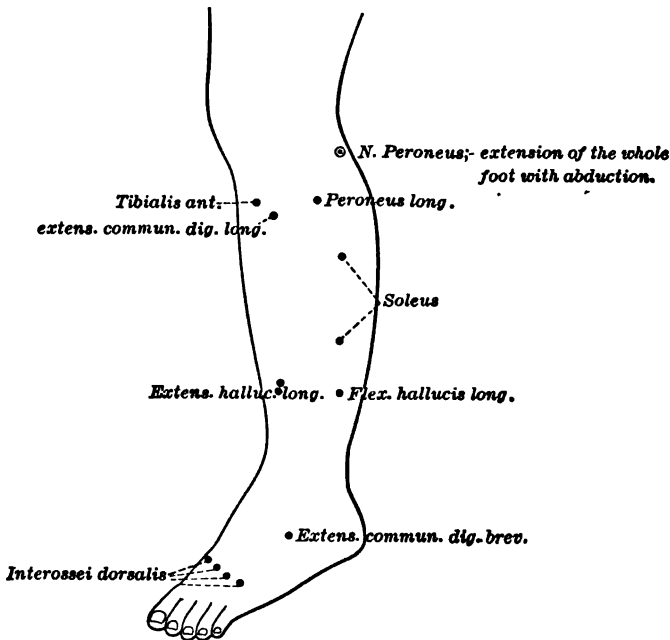


Motor points for the muscles of the anterior surface of the thigh.

Treatment of Paralysis.—After the acute stage is over and the pain and tenderness of the paralyzed parts have subsided to a great extent, it is necessary to begin more active treatment. As the edema of the cord subsides, some ganglion cells are found to be only temporarily damaged, and while we probably cannot hurry their recovery to any extent, we can keep the muscles in good form awaiting that recovery by means of electrical stimulation and massage. But with regard to electrical treatment those muscles which still react to faradism two weeks after the onset of paralysis, should recover their function if properly cared for, while those that have lost their reaction to faradism may or may not recover, and one must form one's opinion of their chances by judging of their reaction to the galvanic current. In any case in treatment it is always better when possible to use the latter current, as it stimulates the muscle fibers directly to contraction, as

well as stimulating through the nerve elements in them, whereas the faradic current, stimulating only through the nerve endings, many fibers of a partially paralyzed muscle would escape stimulation. The accompanying charts of the motor points of the muscles (Figs. 46 to 51) will be found useful in carrying out the treatment with the galvanic current, and in stimulating to contraction individual muscles. Massage and passive movements are necessary both to keep up the nutrition of the muscles and also, and just as important, to prevent contractures in their opponents. Active movements when possible must be encouraged by every means, and it is often possible by having a patient in a bath to obtain voluntary movements of a limb under water long before it is otherwise possible, and a little such encouragement will help very considerably.

FIG. 51



Motor points for the muscles of the anterior surface of the leg.

One must also strive to prevent stretching of a paralyzed muscle either by contracture of its opponents or by allowing it to remain in a faulty position, and one can often fulfil this purpose by arranging an elastic strap to take the place of the paralyzed muscle. Thus in paralysis of the anterior tibial group with foot-drop, an elastic (simple garter elastic) run up from the boot laces to an elastic band about the knee will prevent overstretching of the paralyzed muscles and still will not be so strong as to do away with their action entirely when they begin to regain power.

The tendency toward improvement of the paralysis will continue under treatment for about a year or eighteen months. After this time the condition should come under the orthopedic department and the question of tendon grafting, fixation of joints or braces, must then be considered.

LANDRY'S PARALYSIS (ACUTE ASCENDING PARALYSIS)

This disease was first observed and described by Landry in 1859. He describes it as characterized by an acute ascending paralysis commencing in the legs, spreading to the trunk, and involving the centres for the diaphragm, with as far as he could make out with his more or less imperfect means of investigation no underlying pathological lesions of the spinal cord or peripheral nerves. Since his time numerous cases have been added to the literature which clinically conformed to his description, but as a matter of fact the majority of these should never have been included under this name, as pathological examination showed either a definite multiple neuritis or lesions in the cord of the nature of an ascending myelitis or poliomyelitis, or a combination of both the peripheral and the central lesion. It is probable that had such lesions been present in the cases described by Landry he could have seen them even with the comparatively imperfect means at his disposal at that time, and their inclusion under this heading has only caused hopeless confusion. That Landry's paralysis does exist as a clinical and pathological entity must be admitted when one considers the cases published by Bernard and Westphal, Watson, Omerod, Alber, Morton Prince, Steven and Farquhar Buzzard. In the majority of these cases no abnormality was discovered by pathological examination, but in the more recently examined cases fine changes have been observed when studied by the methods of Nissl and Marchi.

Etiology.—The disease usually affects adult males and it frequently follows acute infections of various kinds. Apparently the disturbances of function of the nervous system are due to a condition of acute intoxication. In many cases no etiological factor can be found. On the other hand, Buzzard has isolated a form of tetracoccus from the blood and the loose vascular tissue forming the external layers of the spinal theca, which when inoculated into the subdural space in a rabbit produced widespread paralysis without any inflammatory lesion of the cord. The germ was recovered from the blood and the theca of the rabbit. MacNamara and Bernstein isolated a similar organism from their non-fatal case, but were unable to produce any positive results by experimental inoculations. The disease sometimes follows exposure to wet and cold.

Symptoms.—Prodromata are usually present in the shape of paresthesiæ in the limbs, pain in the back, and stiffness of the neck. There is, as a rule, some general malaise, but fever may be absent or of a slight degree only. Paralysis of the legs soon follows, flaccid in character, and with loss of tendon-jerks. Very often, as Buzzard has

pointed out, the toes may still be moved even when the rest of the leg is completely paralyzed, unlike what one sees in paralysis due to multiple neuritis. The plantar responses remain of the flexor type as long as they can be obtained. Sensation is not affected; there may, however, be some tenderness of the muscles elicited on deep pressure, and frequently the patient complains of cramp-like pains in the muscles of the paralyzed limbs, which can be relieved, as a rule, by change of position. In the course of a few hours, as in Morton Prince's case, or more generally in the course of a few days, the trunk muscles become involved, the paralysis spreading up the trunk, segment by segment, until the intercostals are all paralyzed and respiration is dependent on the diaphragm and the accessory muscles of respiration alone. The hand muscles may be next involved and gradually the whole arm. The disease may stop here or may progress until the centres for the diaphragm are also picked out and the patient dies from respiratory failure. The pulse may continue of fair strength for some time after respiration has ceased. The intellect remains clear as long as respiration keeps up, and I have seen one patient dying of respiratory failure recover consciousness, recognize his friends, and speak quite rationally when artificial respiration was started, and remain so for some time, so long as artificial breathing was kept up.

Owing to the respiratory weakness from paralysis of the intercostals pulmonary complications are apt to lead up to a fatal issue, but if these are escaped from and the diaphragm does not become paralyzed, the patient will begin to improve. Recovery is slow, but, as a rule, eventually becomes complete. The muscles waste somewhat and the electrical reactions may be impaired, but generally faradic excitability is never completely lost, though it may be much diminished. The muscular wasting, according to Buzzard, is always general and never picks out individual muscles in a limb. Contractures and deformities rarely occur; recurrences are unknown.

Pathology.—No evidence of disease is detectable in the nervous system by the naked eye except a general vascular engorgement. The cord is firm in consistence throughout. Buzzard has reported certain changes distinguishable in the cells of the anterior horn and Clarke's column by Nissl's method and in the fibers by Marchi's stain. Many cells show early pericentral chromatolysis and a few complete or nearly complete loss of chromatin granules and excentration of the nuclei. By the Marchi method the myelin sheaths of the fibers in the cord and to a lesser degree in the peripheral nerves show a diffuse fatty change. This is apparently not nearly so marked as one sees it in true Wallerian degeneration, and the droplets of fat are smaller and more discontinuous; such a condition is not necessarily associated with any loss of function on the part of the axis-cylinders. There is no definite evidence of neuroglial or vessel changes. Enlargement of the spleen, such as is seen in other acute toxic diseases, is very frequent.

Diagnosis.—The differential diagnosis may be one of some difficulty. From acute ascending myelitis the diagnosis is as a rule easy on account

of the sensory paralysis and the involvement of the bladder and bowels. To distinguish between this disease and acute poliomyelitis is more difficult and may be impossible clinically. Buzzard claims that in severe cases of poliomyelitis the constitutional symptoms are more marked, but this is only a matter of degree; the same thing holds good for the paresthesia, pains, and tenderness which are perhaps somewhat more marked in poliomyelitis than in Landry's paralysis. He lays stress on the fact that the escape of any individual muscle or group of muscles, or any marked asymmetry in the paralysis of the two limbs would point rather strongly to poliomyelitis. In multiple neuritis the paresthesiæ are, as a rule, more marked and the deep muscle tenderness very much more evident. The periphery of the limb is more affected than the trunk muscles, and atrophy and electrical changes in the muscles appear very early. Relapses are not infrequent in multiple neuritis while they never occur in Landry's paralysis.

Prognosis.—It is impossible to form an idea of the percentage of mortality in this disease, and one should give an opinion with caution until the progression of the paralysis has definitely ceased. The main fear is pulmonary complications, either pneumonia or bronchitis. Apart from these the outlook for complete recovery is good. Strength returns gradually and steadily, and, as a rule, no sign of the motor paralysis will remain.

Treatment.—In the early acute stages some sort of vapor bath might help in the elimination of toxins. This can easily be arranged so as not to disturb the patient by guiding steam under the blankets of the bed raised a little from the patient by a series of hoops. Rest in the recumbent position with the head slightly raised is essential, and the patient should not be allowed to move himself but should be moved from one position to another at regular intervals, and the limbs made comfortable by a change of position as comfort demands. It is doubtful what good effects, if any, are to be obtained by counterirritation along the spine, and the consequent discomfort of such treatment is sufficient to render it inadvisable. No drug is known which has any effect on the disease process, ergotin and salicylates have been recommended but their utility is very doubtful. Modern research may some day produce some antidote for the toxemia, the underlying cause of the disease. When there is danger of respiratory failure atropine may be of some use in stimulating the centres to tide them over the acute stage; it should be used hypodermically and may be combined with strychnine. The bowels should be opened with a mercurial purge and kept free to avoid any overstraining. With ordinary care there is little danger of bed-sores forming. The bladder may require to be catheterized for the first few days. After the acute stage massage and passive movements of the limbs should be practised daily, combined with stimulation of the muscles, preferably by the galvanic current.

INFLAMMATION OF THE CORD (MYELITIS)

Until recent years there has been a certain amount of confusion surrounding the question of myelitis. Literally the term signifies inflammation of the cord, but practically it has been used to cover any condition of softening and destruction of cord tissue whether due to true inflammation or to occlusion of a spinal vessel, and even when softening of the cord has been caused by pressure, whether by a tumor or by vertebral caries. As in inflammatory lesions elsewhere, myelitis may be of an acute or subacute nature. It is very doubtful whether such a condition as a chronic myelitis exists apart from a form of softening of syphilitic origin, which is discussed elsewhere.

Other varieties are based on the distribution of the disease. Thus when the gray matter is affected the condition is termed poliomyelitis—a sufficiently distinctive disease to claim a chapter for itself. According as the disease involves the whole thickness of the cord at one level, or is scattered indiscriminately, it is termed *transverse* or *disseminated* myelitis. When an extensive area of the cord is involved it is termed *diffuse*, and when due to pressure from spinal caries or extramedullary tumor the term *compression* myelitis is given.

Etiology.—Myelitis, apart from that form of softening of the cord dependent on a syphilitic vascular process, is not very common; it may, however, affect individuals of any age, although it is more commonly met with in patients in middle adult life. In children the poliomyelitic form is more commonly seen. It may be found associated with practically any of the infections and intoxications, more particularly tuberculosis and gonorrhea. It occasionally follows furunculosis, especially when the furuncles are situated close to the spine. It occurs more rarely in measles, variola, scarlet fever, influenza, erysipelas, pneumonia, typhus fever, and following parturition. Overexertion, trauma, and exposure to cold, all seem to have a certain etiological significance, but probably they are only indirectly responsible and the direct responsibility rests on bacterial invasion.

In severe secondary anemias where there has been clinical evidence of an associated subacute combined sclerosis, we not infrequently see a sudden complete paraplegia due to a superadded diffuse myelitis. Injuries to the cord, whether from lacerations, punctured wounds, or hemorrhage into its substance, may set up a definite myelitis and the disease not infrequently follows falls, not necessarily attended with injury to the back, but when a violent effort is made to save one's self. Possibly in these cases some minute injury occurs at the time which forms a focus of lessened resistance to the action of bacteria. I have seen myelitis occur in a man, a painter, who had suffered from lead poisoning of a mild type some years previously, five or six hours after a fall of a few feet in which he had landed on his hands and knees.

Pathological Anatomy.—The meninges of the cord are usually congested over the site of the lesion. The cord itself may show no macro-

scopic change externally, but in some cases, especially if obtained within a few days of the incidence of the disease, the cord over the site of the lesion is slightly enlarged, and has a wrinkled appearance as if the cord were trying to bulge through the interstices of its pia-arachnoid envelope. On palpation through this membrane it feels hard like a pencil, and on section the cut surface swells up above the level of section owing to the congestion and edema of the cord tissue. It may no longer be possible to differentiate the gray from the white matter and the cord substance is of the consistency of cream cheese. When autopsy is obtained, a week or two after the incidence

FIG. 52



Myelitis.

of the lesion, the cord looks smaller at the site of the lesion and on palpation is softer than normal. On section the cord substance is broken down and diffuent in parts, depending on the extent of the destruction. In older cases when sufficient time has elapsed for sclerosis to occur the cord is small, firm, and white at the site of the lesion.

Microscopically in very recent cases there is marked congestion of the vessels, with diffuse leukocytic infiltration, most marked about the vessels, numerous small hemorrhagic extravasations are frequent, and there is indiscriminate destruction of the parenchymatous tissues affecting both the cells in the gray matter and the fibers in the white.

The myelin sheaths and axis-cylinders are broken up into granular particles which are later absorbed. Many of the anterior horn cells may be found to have already disappeared and others will show

destructive changes of different grades varying from mere chromatolytic changes to a condition of granulation with the nucleus swollen and indistinct; later the nucleus disappears, and the cell processes break up, and a mere shadow of the cell is left which in its turn is soon absorbed. Compound granular cells are numerous, containing fatty granules, the products of the destruction. Toward the edge of the lesion the evidences of destruction gradually diminish, and the changes in the nerve cells and fibers are less marked. The axis-cylinders appear somewhat swollen and the myelin sheaths thinned and distended. Near the site of the active focus of destruction many lacunæ will be seen where the nerve fibers have become absorbed, but the neuroglial tissue ground-work has not been completely demoralized. The neuroglia in time proliferates, and in old-standing cases a regular scar is formed with increase in the neuroglia, and numerous "spider cells" may be seen.

Above the lesion one gets a secondary degeneration in such ascending tracts as may have been involved in the destruction, and similarly, below this level, the descending tracts show degenerative changes.

Myelitis may be directly associated with an inflammation of the meninges, a meningomyelitis; frequently this is of syphilitic origin, but may also occur in connection with inflammatory conditions of adjacent parts, and is occasionally seen in the various cerebrospinal meningitides.

Transverse myelitis, when the complete cross-section of the cord is involved at a certain level, most commonly occurs in the dorsal region, probably because of peculiarities and relative insufficiency of the vascular supply in this part. In this form there is usually the one area of the cord affected. In the disseminated form, small areas of inflammation are scattered more or less indiscriminately throughout the nervous system, and not infrequently the optic nerves are affected, giving rise to a retrobulbar neuritis which later progresses to partial atrophy of the optic nerve.

Marinesco has shown that bacteria disappear from the cord in a few days and, as a general rule, cultures from myelitic foci are negative, although staphylococci, streptococci, and pneumococci have all been frequently found associated with the lesion. Experiments have shown, however, that not only bacterial inoculation but inoculation of toxins from bacterial growth might produce the lesion, and clinically many cases seem to be of this nature. Sir William Gowers suggests the probability that many cases of myelitis are due to a thrombosis of the finer spinal bloodvessels, pointing out that the initial lesion may quite disappear in the intense inflammation which it excites. This is no doubt a very common process in syphilitic cases, but is probably very rare apart from this disease save in those cases where the meninges are primarily involved and the cord lesion is only secondary.

Symptoms.—The typical form of myelitis is the transverse variety and the most common site of affection, as has already been stated, is the dorsal cord. The symptoms are those of a transverse lesion of

the cord of acute onset. Prodromal symptoms, such a general malaise, shivering, loss of appetite, may be present for some hours or days, or may be altogether absent. Pain in the back is sometimes complained of, but regular girdle root pain is seldom present. Numbness with heaviness and motor weakness of the legs appears and rapidly progresses to a more or less complete paraplegia with spasticity more or less marked. The paralysis may reach its height in anywhere from a few hours to a few days. In cases where the onset is rapid, one frequently finds a flaccidity or loss of tone in the paralyzed extremities, for the first few days at least, and this is to be accounted for by the sudden irritative destruction of the pyramidal tracts increasing their inhibitive action on the lower motor neurones. For as the irritation ceases and degeneration sets in, removing the inhibitory action of these fibers, the tone of the muscles in the paralyzed extremity increases and soon a definite spasticity develops with increased tendon-jerks. This may become very severe so that it is almost impossible to bend the legs passively; in such cases, if neglected, contractures develop in the course of time with consequent deformity. When contractures have developed it may be impossible to obtain the tendon-jerks. Not infrequently the patient suffers greatly from spasmodic twitchings of the legs of a reflex nature, preventing sleep. In other cases the paraplegia may not be quite so complete and the patient can move his legs a little but with no strength—possibly he can move the toes only—depending on the extent of the lesion. The extent of the paralysis will depend on the level of the lesion in the cord; if the lower cervical region is affected the muscles of the arms will show a flaccid atrophic paralysis with loss of tendon-jerks, respiration will depend on the diaphragm, while the legs will be paralyzed and spastic, the bladder also will be paralyzed. If the upper cervical segments are involved the consequent diaphragmatic paralysis will cause death by asphyxia.

Below the level of the lesion there will be loss of sensibility more or less marked to all forms of stimulation, and the reader is referred to the charts showing the segmentary sensory distribution in the skin. The tendon-jerks are all exaggerated below the level of the lesion and both rectus and ankle clonus can usually be obtained. The plantar response is practically invariably extensor in type (Babinski's phenomenon) from the first; Oppenheim's tibialis phenomenon may also be positive. The abdominal reflexes are lost below the level of distribution of the lesion and may be present above it if the injury is between the seventh and twelfth dorsal segments, and in this case also Bevor's sign will be present, that is, if the patient be told to rise into the sitting position from the recumbent while his arms are folded, on making the attempt the umbilicus will be seen to ride upward toward the ribs, owing to the contraction of the non-paralyzed upper portion of the recti abdominis and the stretching of the paralyzed lower portion.

The bladder being cut off from all central control will act reflexly, giving rise to retention of urine, with possibly the incontinence of

overflow, rendering catheterization necessary. This very often goes on to a complete incontinence later. The bowels are affected similarly. Trophic disturbances are very apt to affect the skin over the paralyzed parts exposed to pressure, and bed-sores form over the sacrum, the trochanters, and on the heels, often with great rapidity. The incontinence of urine and feces of course adds to this tendency considerably.

The picture will vary somewhat according to the site of the lesion and its extent. If the lumbar and sacral region be affected there will of course result an atrophic flaccid paralysis of the lower extremities with loss of tendon-jerks. Or if the focus be confined to one-half the transverse diameter of the cord, there will result a Brown-Séquard type of paralysis, with the spastic motor paralysis and loss of sense of position in the homolateral extremities below the level of the lesion, and the loss of sensibility of the opposite extremities, especially to heat, cold, and painful stimuli.

In disseminated myelitis the symptoms will vary with the site of the lesions, and these are not confined to the cord but may affect the pons, medulla, or any part of the brain. The disease not infrequently follows influenza and the exanthems. The onset is febrile in character with general malaise, headache, and chilly sensations, the picture of a toxic condition, and in the course of a few hours there will be evidence of organic disease of the nervous system—perhaps a complete paraplegia, or one leg may be paralyzed completely, the other only incompletely. Possibly paralysis in the area of distribution of some cranial nerve may be the only evidence, or it may be involved at the same time as the extremities. In a large percentage of cases the optic nerve is affected giving rise to a retrobulbar neuritis. Sometimes one sees a more or less complete hemiplegia. In other cases a flaccid paralysis of an extremity with loss of reflexes. Impairment of sensibility will depend on the parts affected. As a rule the extensor plantar responses give evidence of involvement of the pyramidal tracts. Owing to their long course through the brain and cord we could hardly expect them to escape being caught in one or other of the scattered lesions of the disease.

Usually after a few days the fever subsides and the patient feels very much better, and gradually in a few weeks the paralysis clears up to a greater or lesser extent, although never completely. If the disease has been mild there may be only a certain difficulty in walking, especially when fatigued—perhaps affecting one leg more than another, and possibly some impairment of vision. Examination will then show practically nothing but bilateral extensor plantar responses and a partial optic atrophy. Of course the symptoms will vary with the extent and situation of the lesions, and we sometimes see intention tremor of the upper extremities, nystagmus, and spasticity of the legs. Practically speaking, it is usually impossible clinically to distinguish between disseminated myelitis and disseminated sclerosis.

Course and Prognosis.—Both these depend on the site and the severity of the lesion. When the upper cervical cord is affected with

consequent respiratory paralysis death of course follows rapidly. In other cases, death when it occurs is due to other complications such as cystitis with an ascending infection of the urinary tract and general pyemia. Pneumonia is not an uncommon terminal event in these cases, or frequently, septicemia from bed-sores may lead to a fatal termination. But, as a rule, especially in cases of acute onset, while the initial paralysis is severe, it clears up to some extent fairly quickly. Some cases make an almost complete recovery, but usually some permanent paralysis is left. In my experience the prognosis, speaking generally, is better in cases in which the onset has been acute than in those in which the symptoms have taken some days to become complete.

Treatment.—The most common cause of death in these cases is from cystitis and bedsores, therefore our first aim must be to prevent these. The patient should be put on a water- or air-bed immediately, air-cushions under the buttocks and padded rings for the heels are in my experience but poor substitutes for the water-bed. Scrupulous care must be taken of the back, and the skin should be rubbed thoroughly and systematically with alcohol twice a day, the sheets should be kept clean, and it is well to dust them with oxide of zinc and starch powder. The draw-sheet should be changed as occasion demands; the incontinence of feces and urine render this very frequently necessary. If the skin becomes reddened and shows signs of breaking down a lotion made up of 10 grains of tannin to the ounce of rectified spirits, or a solution of alum, is often useful in preventing the actual formation of a sore. If the skin becomes broken and an actual bed-sore form, it should be dressed with zinc oxide ointment, or a dusting powder of aristol if it remain clean. A septic sloughing bed-sore should be swabbed out thoroughly with hydrogen peroxide or some other aseptic solution.

Catheterization is usually necessary for some time and scrupulous asepsis must be used. It is good practice to administer some urinary antiseptic by mouth, for example urotropin, grains v to x, three times a day. The bladder should not be allowed to become overdistended, but should be emptied every eight to ten hours. A soft rubber catheter, rendered perfectly aseptic by boiling, is best, and the orifice of the urethra should be sponged with some mild antiseptic solution before this is passed. The catheter is usually lubricated with sterilized vaseline. If cystitis occur in spite of these precautions, and it sometimes will, especially where the bladder wall has been allowed to become overdistended, the bladder should be washed out daily with a warm solution of boracic acid—10 grains to the ounce.

The involuntary spasmodic twitchings of the legs are often very troublesome, wakening the patient from sleep or preventing his getting to sleep; they are best controlled by bromides or small doses of hyoscine hydrobromate. Hot applications, and when it is possible hot baths, are also often useful in affording relief from this symptom, and also from the great spasticity of the lower extremities.

The patient's position in bed should be frequently changed, not

only to prevent the formation of bed-sores but to avoid the tendency to hypostatic congestion of the lungs when the intercostal muscles are paralyzed.

At the onset of the disease a calomel purge will be indicated, followed by a mild saline and again by mild diaphoretic treatment for the elimination of toxins from the system. This is probably best done by following Oppenheim's suggestion of introducing steam by a suitable apparatus under the bedclothes.

As recovery begins to take place general massage will be very helpful and passive movements of the paralyzed extremities will prevent contractures forming. In fact, from the beginning one should endeavor by all means to prevent the formation of deformities—cages may be used to keep the weight of the clothes off the feet, and supports, as sandbags or a cross-board at the foot of the bed, will also aid in preventing foot drop. Something of the nature of a Gowers' boot can often be devised by cutting a large piece of leather so that it can be bent to fit around the leg and foot, carefully padded, and laced up. An elastic should now be run from near the toe and attached to the upper part of the boot just below the knee. In this way the foot can be maintained at a right angle to the leg.

SPINAL GLIOMATOSIS AND SYRINGOMYELIA

(*σῦριγξ* = a flute or pipe, and *μυελος* = marrow).

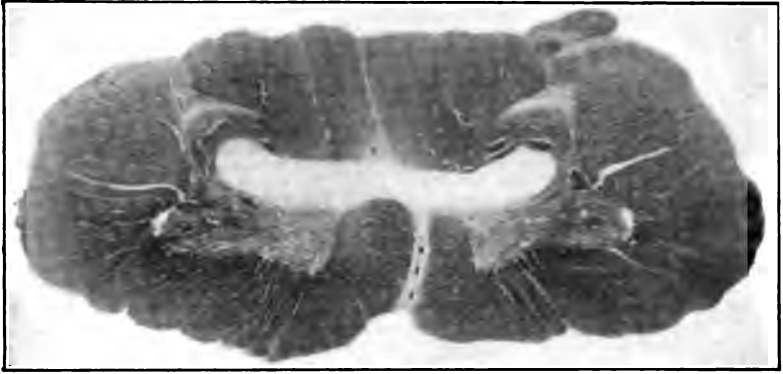
These two conditions will be considered together, as usually they are associated the one with the other and the symptoms produced are identical. In spinal gliomatosis, or gliosis, we have an overgrowth of neuroglial tissue in the centre of the cord, which, while not giving rise to any great enlargement of the cord locally, forms a definite tumor growth in parts, while at other levels it breaks down and forms cavities in the long axis of the organ.

There is also another condition which should be mentioned in this place, namely, a simple enlargement of the central canal, *hydromyelia*, which may be either congenital and in such cases may be associated with other evidences of retarded development, as spina bifida and such like deformities, or may be due to an oversecretion of the ependymal epithelium and associated or comparable with the condition of hydrocephalus.

Etiology.—One can realize that such cavities in the lumen of the cord might also follow hemorrhage into its substance (hematomyelia), and it has been held by some that this cavity formation may follow foci of inflammation of the cord (myelitis). While any of these various causes may be active in individual cases, the general belief now is that the great majority of cases are caused by defects in the developmental processes in the closing in the neural canal, so that nests of glia cells being cut off, lie latent until some exciting cause starts them to grow and proliferate. Thus a primary gliosis is formed about the

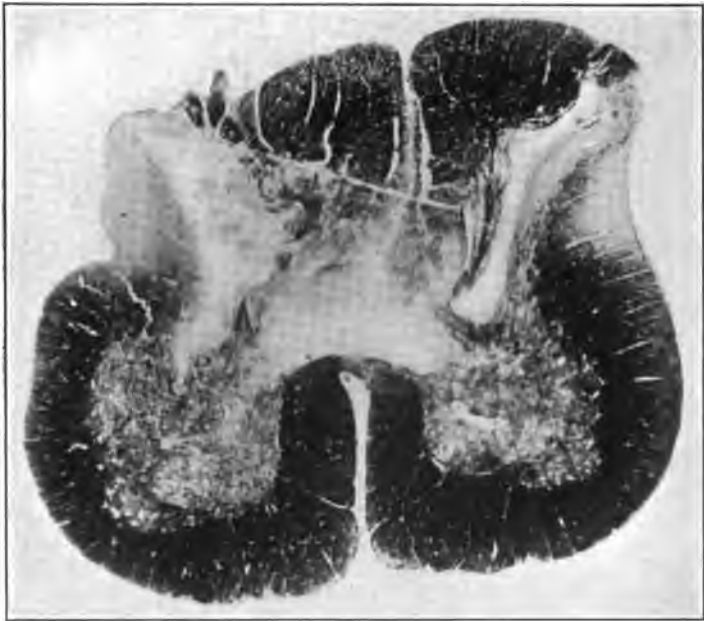
PLATE XLIII

Fig. 1



Syringomyelic Cavity in the Cord (Cervical Region).

Fig. 2



Syringomyelic Cavities in the Cord (Lumbar Region).

1900

PLATE XLIV

Fig. 1



Another Level of the Cord, showing Gliomatous Tumor.

Fig. 2



Syringomyelia, showing the Curvature of the Back and the Atrophy of the Small Muscles of the Hands.

centre of the cord which may later break down giving rise to cavity formation. So frequently has it been observed that the onset of this disease seems to follow some injury to the back, that it appears possible that trauma may act as an exciting cause in starting the proliferation of these nests of embryonic glial cells. The conditions in which cavity formation occur in the cord are known as syringomyelia.

Pathological Anatomy.—On exposing the cord one may observe some little fulness in some parts while in others its appearance is broadened and flattened, and possibly it may be thrown into longitudinal folds, especially if the fluid has drained out of the cavity. On palpation the cord feels like a tube under the finger while at other levels it may be firmer than normal. On section, at one part the centre of the cord may have a firm pale yellowish colored core, while at other levels definite cavities exist containing a clear fluid. The cavity may be of any size and any shape but is usually situated about the posterior part of the gray commissure. It is usually surrounded by a more or less thick margin of a homogeneous pale yellowish color, the proliferated neuroglial tissue. The cavity may be distended with fluid and so give rise to an involvement of the pyramidal tracts by pressure, or the actual proliferation of the glial tissue may involve these or the anterior horns of the gray matter. This gliosis may extend the whole length of the cord, or may be localized more or less to one part. The cervical region is more commonly affected but cases have been reported where the neoplasm extended upward to the fourth ventricle and even to the internal capsule. Not infrequently there will be found at one level a double cavity, one in each posterior horn; but if these be followed up they will be found to unite into one cavity or into a single mass of glial tissue. (Plates XLIII and XLIV.)

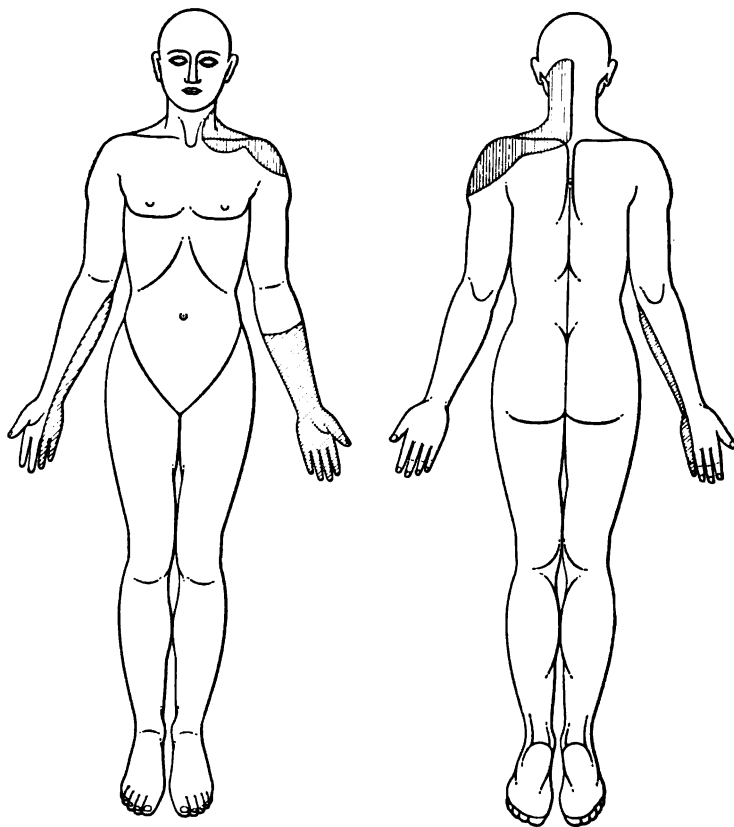
In the condition known as hydromyelia, where there is simply an enlargement of the spinal canal, the margins of the cavity will be lined more or less completely with cuboidal epithelium.

Microscopically one finds the neoplasm is made up of glia cells and fibers in varying proportions.

Symptoms.—The symptoms will vary to some extent according to the site of the disease. As this is most commonly situated mainly in the cervical enlargement and the dorsal cord, it will be well to take up first a typical case. In any case there will be muscular atrophy and a peculiar loss of sensibility, and to this may be added evidence of a lesion of the pyramidal tract and very often vasomotor and trophic disturbances. In the typical case one finds a progressive atrophy of the muscles of the upper extremities and shoulder girdle and very often of the spinal musculature as well with consequent spinal curvature. There may be contractures of the unopposed healthy muscles of the arms with the formation of deformities. (Plate XLIV, Fig. 2.) Fibrillary twitchings are usually to be seen in the muscles when they are first affected. The electrical reactions of the muscles will depend on the amount of destruction of the cells in the anterior horns supplying those muscles. In some there will be the characteristic reaction of degeneration, in

others simply a diminution in their reaction to the various forms of electrical stimulation. In the lower extremities there is, in well-developed cases, invariably all the evidences of a lesion of the pyramidal tract. The patient in walking has difficulty in raising his toes off the floor, he wears out the soles of his shoes at the toes; there is stiffness in the legs due to the increase of tone in the muscles. The knee- and ankle-jerks are exaggerated and usually marked ankle clonus is obtain-

FIG. 53



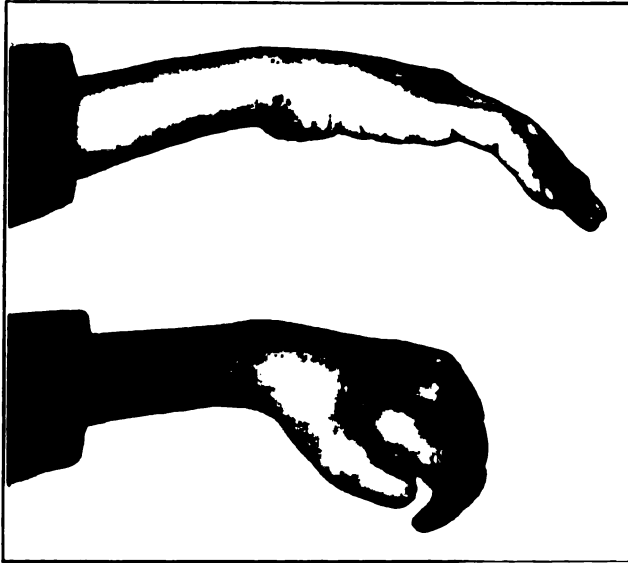
Syringomyelia, showing the disassociated loss of sensibility. Areas of loss of sensibility to pain.

able. The plantar responses are of the extensor type (Babinski's phenomenon) and the abdominal and epigastric reflexes will in the majority of cases be unobtainable.

So far there is nothing to distinguish the condition from one of amyotrophic lateral sclerosis with which it was confused clinically for many years. But in syringomyelia we will find also peculiar and characteristic disturbances of sensibility to pain, heat, and cold, of a dissociative type, that is, the area of loss to pain will not coincide absolutely with

PLATE XLV

Fig. 1



Syringomyelia. Trophic Disturbances
"La Main Succulente."

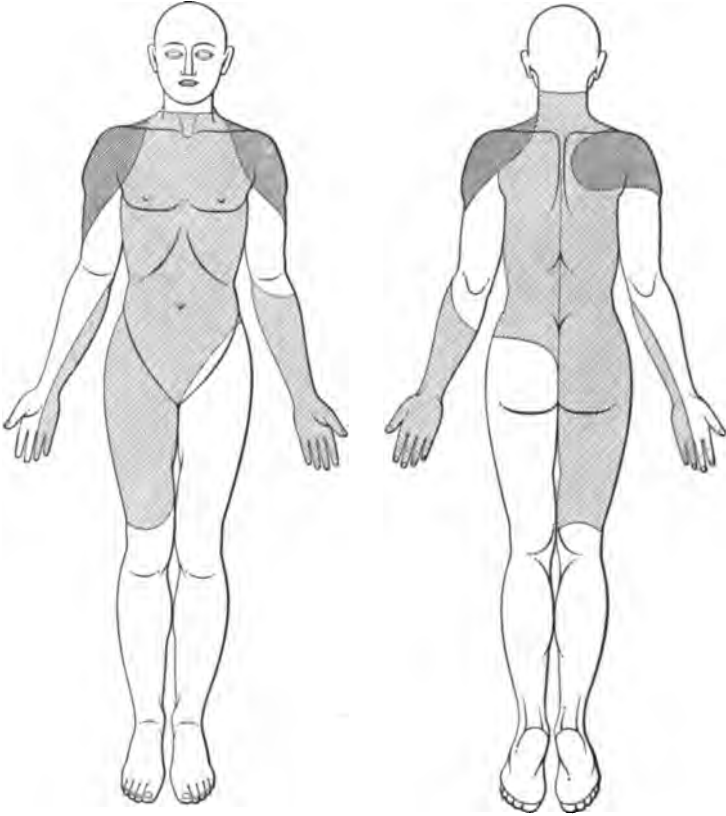
Fig. 2



Syringomyelia. "Prediger Hände."

that for heat, or neither will coincide exactly with that for sensibility to cold (Figs. 53 to 55). These areas of loss of sensibility do not correspond to the peripheral nerve distribution but follow rather the segmental type. Tactile sensibility is, as a rule, unimpaired save in old standing and very advanced cases. In rare cases the sense of position of the muscles may be impaired, giving rise to an ataxia—often confined to one lower extremity.

FIG. 54

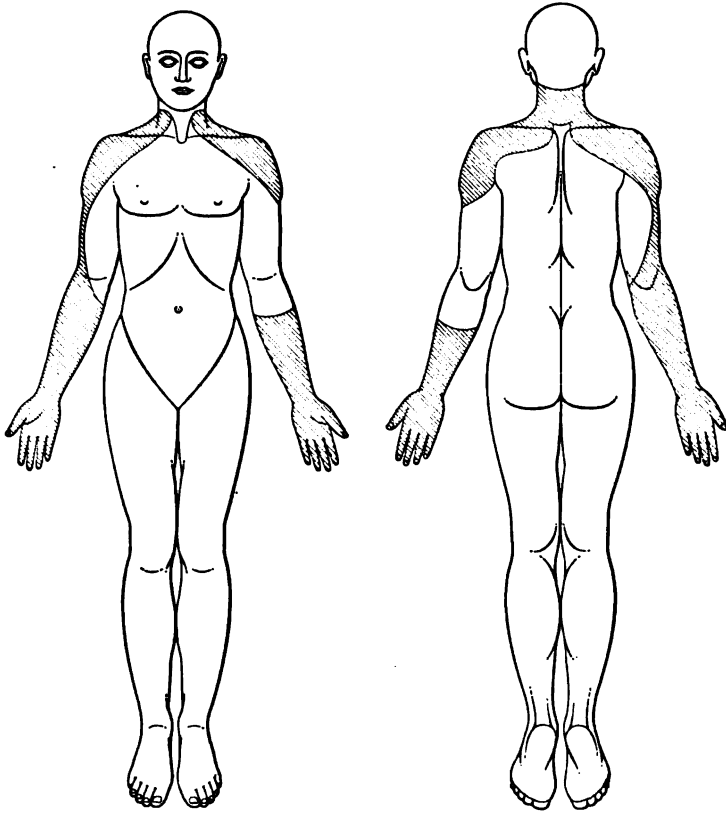


Syringomyelia. Areas of loss of sensibility to heat.

Trophic disturbances are quite frequent and varied. A hand may become much thickened and swollen on its dorsal surface but does not pit on pressure (main succulente of Marinesco). (Plate XLV, Fig. 1.) The skin on the back of the hand is quite red, while on the swollen spindle-shaped fingers the skin is smooth and glossy and when cold of a reddish-blue color. Whitlows are a frequent complication and often appear in close succession in some cases. They are quite painless, as a rule, and on this account are apt to be quite dangerous. It is only when from the extent of the surrounding inflammation, tissues are involved

that still retain their sensibility, that pain is complained of. Owing to the loss of trophic control of the tissues suppuration spreads quickly, and frequently permanent damage and deformity are caused very rapidly. Trophic disturbances may also affect the bones or the skin, and various disturbances of the sweat secretion have been described. Morvan's type of the disease is characterized by repeated affections of the affected parts with ulceration and necrosis. The clinical picture

FIG. 55



Syringomyelia. Areas of loss of sensibility to cold.

varies somewhat with the site of the lesion in the cord. Thus one may get the parts supplied by the bulbar nuclei affected, with atrophic paralysis of the tongue and mouth muscles, a condition of syringobulbia; or the lumbosacral cord may be affected with the consequent atrophic paralysis of the parts supplied.

The onset of the disease is so gradual that usually when the patient comes under observation between the ages of twenty-five and forty years for some trophic disturbance of the muscles, skin, or joints the

characteristic symptoms of the disease are well marked. The course is, as a rule, steadily progressive, although remissions may occur. Death usually occurs from some complication, either cystitis or septicemia, from some necrotic area, or by involvement of the vital centres in the medulla.

Diagnosis.—As a rule this is not difficult. The characteristic loss of sensibility will distinguish the disease from amyotrophic lateral sclerosis or from progressive muscular atrophy. Brachial neuritis may give rise to atrophy of the hand muscles and to loss of sensibility, but this usually is to all forms of sensibility, and corresponds in extent to the nerves affected. Of course in neuritis the pyramidal tracts would not be involved and there would be no signs of spastic paraplegia.

Prognosis.—This is always unfavorable. Nothing can be done to influence the disease process, although remissions occasionally may occur. The course is, as a rule, very slowly progressive and it is usually many years before the patient becomes absolutely incapacitated.

Treatment.—Although we can do nothing to influence the disease process much can be done for the associated conditions. The patient should be warned of the great danger attending wounds, however insignificant, when situated in analgesic parts, and when these do occur they should receive immediate attention and special care to prevent infection. Should infection take place surgical intervention with proper drainage and suitable dressings should be immediately requisitioned. In the early stages massage and passive movements may improve temporarily the strength of the weakened muscles and lessen the spasticity of the lower extremities. The muscular spasm of the lower extremities may be relieved by bromides, or when these fail by hyoscine hydrobromate.

HEMATOMYELIA

Etiology.—Hemorrhage into the spinal cord may occur, as has been already mentioned, into a syringomyelic cavity or gliosis, without any other apparent extraneous etiological factor being present. It occurs probably far more frequently, however, as the result of trauma, either when there is associated fracture or dislocation of the vertebræ, or indeed in many cases when there has been no direct blow to the back, but when the individual has sustained a hard fall, landing on the feet or even on the buttocks, or as not infrequently happens, when a person diving finds the water not as deep as anticipated and strikes the bottom with his head. It may occur also as the result of great muscular exertion, and cases are reported where it has come on while the patient was carrying a heavy weight. I have seen it produced in an otherwise apparently healthy woman, by fright combined with sudden great muscular exertion in escaping from a runaway horse, with resulting sudden complete paraplegia. It may occur as a result of excessive coitus. Difficult labor is a frequent cause of hematomyelia in the newborn child. Hemorrhages into the cord occur also in acute

poliomyelitis and other acute inflammatory conditions, and in patients who have died in convulsions, when the respiratory muscles have been affected, and there has been a somewhat rapid asphyxia. But these must not be included as cases of hematomyelia since the symptoms are all of the primary diseases. This disease is far more common in males than in females, probably altogether because of the difference in their occupation and the greater exposure to accidents in the case of men. It may occur at any age, but is most frequently met with between the ages of twenty and forty.

Hemorrhage into the cord almost always affects the gray matter, partly because this part is more vascular and partly because here the vessel walls have less support from the cord tissue. The effusion may involve both horns, or may be confined to one side. In rare cases only is the hemorrhage severe enough to burst through the cord substance into the spinal canal or rather into the membranes. It rather spreads lengthwise in the cord as the direction of lesser resistance and in some cases may extend quite a distance. Not infrequently numerous minute lesions occur scattered throughout the cord.

Symptoms.—The onset is invariably sudden and without warning, but there may be a gradual increase in the symptoms for one or two hours or more, due to the continuation of the hemorrhage and also to the consequent surrounding edema affecting neighboring structures. The symptoms will vary with the site of the lesion, but, as a rule, there is more or less complete paraplegia with sensory loss below the site of the injury. The bladder functions are disturbed giving rise to retention of urine, necessitating catheterization. Vasomotor and trophic disturbances may come on very rapidly. The lumbar enlargement is frequently the site of such a hemorrhage, with the subsequent flaccid paralysis of the muscles of the legs and loss of knee- and ankle-jerk and later atrophy of the muscles may develop. There will, of course, be loss of sensibility of the segmental type. When the cervical enlargement is affected, the muscles of the arms or hands will be more or less paralyzed and flaccid, while the legs will show a paresis with increase in the tone of the muscles and the knee- and ankle-jerks will be exaggerated, due to the sudden pressure on the corticospinal tracts. The paralysis and contractures may give rise to an appearance described by Oppenheim as the *Prediger Hände*, or preacher's hand, as in Plate XLV, Fig. 2. Not infrequently such a hemorrhage is confined to one side of the cord in the gray matter and one gets all the evidences of a Brown-Séquard paralysis with flaccid paralysis of one arm and a spastic paralysis of the leg on the same side, with a loss of sensibility to pain, heat, and cold on the opposite side. The sense of position in the muscles of the spastic leg may be impaired as well. Pains in the area of distribution in the nerve roots may be present from the first. Death seldom follows uncomplicated hemorrhage into the cord unless the upper cervical segments are severely affected, and, as a rule, with proper care the patient soon shows signs of improvement even within a few days. As the effects of the shock and disturbances of pressure wear off

the paralysis, both sensory and motor, diminishes and control of the bladder is regained, and this improvement is, as a rule, steadily progressive. Usually this improvement only goes a certain way toward recovery, and some sign of the lesion remains permanently. Frequently this is seen in an atrophic paralysis of certain muscles; as has been said the lesion generally originates in the gray matter of the cord.

Differential Diagnosis.—The suddenness of the onset of motor and usually sensory paralysis with pain in the back and in the area of distribution of the nerve roots is the characteristic feature of this disease. It is to be distinguished from hemorrhage into the meninges by the absence of the great rigidity of the paralyzed muscles which is seen in the latter condition. Lumbar puncture, with withdrawal of the cerebrospinal fluid, would of course immediately show whether or not there has been extravasation of blood into the meninges. The absence of prodromal symptoms is characteristic. In myelitis there is usually some warning, the onset is slower and, as a rule, there is fever. In thrombosis of the spinal vessels with softening there may be no fever but the onset is much slower and warnings, as a rule, are felt for some time before the onset of the paralysis. Acute poliomyelitis is usually associated with some preceding fever and general malaise. Some cases do occur in which the child, while playing around, falls, apparently as the first evidence of the onset of the paralysis, and in such cases if there is no further evidence later of inflammatory foci in other parts of the cord it would be difficult to exclude a diagnosis of hematomyelia, save that with fairly extensive motor paralysis there would be no sensory impairment or signs of involvement of the pyramidal tracts present in poliomyelitis as one would expect to find in paralysis due to hemorrhage.

Treatment.—Rest in bed is absolutely essential and should be obtained as soon as possible; ice-bags applied along the spine over the site of the hemorrhage may aid in stopping the extravasation. All exertion should be forbidden, and the patient should only be moved with great care and sufficient help to insure his not attempting to help himself. The bowels should be well opened and kept loose to prevent any straining. If the paralysis is severe it would be well to get the patient on a water- or air-bed as soon as possible to prevent the formation of bed-sores. The retention of urine will necessitate catheterization at least for a while and the usual aseptic precautions are much more essential than in the ordinary surgical case. It is advisable to put the patient on urotropin, grain v or x, three times a day, to lessen this tendency to cystitis. After the acute stage is over and recovery has progressed well, warm baths and massage with passive movements are indicated for the spasticity, and electrical treatment with the galvanic current to the atrophic muscles. It is well to keep the patient in the recumbent position for some weeks to prevent any chance of a recurrence, and when he first begins to walk, some sort of a wheeled carriage on which he can rest his arms and support the weight of his body, while he uses the legs, will be found extremely helpful.

TUMORS OF THE SPINAL CORD AND ITS MEMBRANES

Since 1887, when Horsley first operated successfully on a case of spinal cord tumor, diagnosticated and localized by Gowers, the interest in such neoplasms has increased materially and such operations are by no means uncommon. Tumors of the cord can be classified into those of the coverings of the cord, and secondly, those of the cord itself. The first may be divided again into those which originate in the bony covering of the cord or from the softer tissues about the vertebral bodies, and involve the cord secondarily—*vertebral tumors*, and secondly those which begin in the membranes, either the dura or the pia arachnoid—*intravertebral tumors*. These latter may again be divided into intra- and extradural tumors. Tumors originating in the cord itself are termed *intramedullary tumors*.

Between these two groups we must consider those neoplasms which, commencing in the pia-arachnoid, spread in and invade the cord itself. Most frequent and of most practical interest are the meningeal tumors, if we except syphilitic gummata and gliosis spinalis, which are discussed under their separate chapters. Spinal gummata will of course cause the same symptoms as any other tumor of the cord, but practically from the point of view of treatment especially it is very different. Spinal caries also will be taken up separately.

Vertebral Tumors.—Symptoms.—Vertebral tumors give rise to symptoms referable to the nervous system by gradual involvement of the spinal roots and the cord itself; they are of a carcinomatous or sarcomatous nature as a rule—sometimes bony exostoses act in the same way. Carcinoma of the vertebræ is almost always secondary to a focus elsewhere, it may be in the stomach, uterus, or especially frequently in the breast. The consequent destruction of the vertebral bodies gives rise to a gibbus formation and squeezing of the cord or nerve roots. Sarcoma and osteosarcoma usually originate in the periosteum or in the marrow of the vertebral bodies, especially in the pelvic bones, in which case they give rise to pressure symptoms on the sacral plexus. In other cases by extension through the intervertebral spaces they may cause pressure directly on the cord itself. Bony exostoses cause pressure on the cord or roots by their growth from the inner surface of the vertebræ or from their laminæ. One should in this place mention aneurysms of the aorta, which by gradual erosion of the bodies of the vertebræ may give rise to pressure symptoms, generally of the nerve of roots with consequent girdle pains. The patient usually complains of severe boring pains in the spine, especially on movement, and stiffness of the back, and frequently of neuralgic pain in the region of the intercostal nerves due to their compression, and finally the cord itself may become compressed. Severe intercostal neuralgia with a history of periods of intense pain varying with periods of comparative ease, should make one think of aneurysm of the aorta pressing on the intercostal nerves. Arthritis deformans should also be considered here,

as it not infrequently affects the vertebræ and may cause root pains by involvement of the nerve roots as they pass out through the intervertebral foraminæ, it practically never leads to pressure on the cord itself. Severe pains and stiffness of the vertebral column are the usual results.

In tumors of the vertebræ, as carcinoma and sarcoma, the bone itself is first involved and then the spinal roots and finally the cord itself begins to be compressed. It may be simply compressed or softening may occur as the result of cutting off the blood supply. It seldom happens that it is directly infiltrated by the newgrowth, the dura being as a rule sufficient protection.

The symptoms produced are the same for any slowly progressing pressure on the cord and its roots, and one must differentiate between these neoplasms and a condition of caries of the vertebræ. First, there are the evidences pointing to disease of the vertebræ and in some cases these are insignificant or possibly absent altogether, so that one is inclined to think of a neuritis. When they are present there is localized pain, especially on movement or exaggerated by any jar of the body. The patient usually holds the affected part of the back stiff, especially in turning. Localized tenderness on deep pressure over the spinous processes can be elicited. Often one can make out enlargement of the bone itself, especially in sarcomata. The involvement of the nerve roots makes itself evident by pain of a persisting character, like a severe neuralgia. Their location depends naturally on the site of the lesion, but they are almost always bilateral. It may be that one side is first involved and the other shortly after. Bilateral sciatica in elderly people should make one think of carcinoma of the vertebral column. Sometimes there is a hyperesthesia in the region of the nerve affected; seldom is a definite anesthesia produced until the later stages. Herpes zoster is frequently seen. Spasms and cramps may effect the individual muscles supplied by the nerves involved, with later a definite paresis or paralysis of an atrophic character with the usual electrical disturbances.

Once the cord is definitely involved, either slowly by pressure or rapidly by being squeezed in the sudden gibbus formation, or by softening due to the involvement of its blood supply, we get a paraplegic condition and loss of sensibility below the level of the lesion with disturbances of the bladder and bowel functions. In some cases the involvement of the cord may be only on one side producing a more or less typical Brown-Séquard paralysis.

Diagnosis.—The diagnosis of a vertebral tumor is, as a rule, very easy. Vertebral caries, however, may lead to a very similar picture and the points of differentiation are often vague and uncertain. Caries is usually a disease of earlier years, but not always; as a rule there is evidence of tuberculosis elsewhere. Carcinoma is usually secondary to carcinoma in other parts. In simple intercostal neuralgia the site of the pain is by no means so definite, the pain is not so urgent, and it is not exaggerated to such an extent by movement. From intraverte-

bral tumors the differentiation may be made on considering the course of the disease and the succession of the symptoms. In vertebral tumors one sees first, at least in typical cases, the pain and stiffness in the spine, following this the root symptoms appear, and later still the evidence of pressure on the cord itself. While in intravertebral tumors the signs of pressure on the cord and the nerve roots are the first symptoms and only later, if at all, is there any evidence of tenderness of the vertebræ to pressure or percussion, or any pain or rigidity on movement. Intravertebral tumors practically never involve the spinal column, therefore gibbus formation rarely results from such a neoplasm. In vertebral tumors the pains are, as a rule, bilateral from the beginning, while in meningeal tumors, as we will see, they are usually confined to one side, for a long period of time at least.

The diagnosis of a vertebral tumor having been made it is necessary to form an opinion as to its nature. The presence of carcinoma elsewhere, or the history of having had such a tumor removed, would be strong evidence in favor of its being of a similar nature. The form and extent of a sarcomatous tumor are, as a rule, easy to recognize; these two forms are by far the most commonly met with. Bony exostosis also will be easily recognized by the aid of the skiagraph, besides which there will often be evidence of the same condition in other parts of the skeleton.

Prognosis.—The prognosis in vertebral tumor is naturally bad, especially so when they are of the nature of carcinoma or sarcoma. Nothing that we can do will prevent the spread of the disease and the progressive involvement of the tissues. The duration of the disease varies and a fatal termination usually is due to some complication or as a direct result of the affection. Bed-sores, cystitis, and pyelitis usually occur, especially if the lumbar enlargement be involved, whereas if the upper cervical region be affected respiratory failure may be the direct result of involvement of the centres. Surgical interference while it can give no hope of curing the condition is often indicated when the pain from pressure on the nerve roots is very severe. In such a case it would be advisable to cut such posterior roots as are involved in order to relieve the pain and render the patient less uncomfortable. Morphine in such cases is naturally absolutely indicated.

Spinal Cord Tumors Developing Inside the Vertebral Canal.—We must now consider the true spinal cord tumors developing inside the vertebral canal—these may develop in connection with the cord itself or may spring from its meningeal coverings. The latter are the more frequent and also of more practical interest. They may be either intra- or extradural, the former are probably somewhat more common. They are usually of the nature of a primary tumor, although occasionally metastatic tumors do occur. Not infrequently the simultaneous development of similar tumors in other organs is observed.

Extradural Tumors.—Extradural tumors may be of the nature of a lipoma developing in the extradural fatty tissue, or again sarcomas may develop from the outer surface of the dural membrane, or the

periosteum of the inner surface of the vertebræ, the latter belong rather to the vertebral tumors. Echinococcus cysts are not uncommon in certain localities and are usually situated between the dura and the spine; often they are multiple. Metastatic growths, as carcinoma or sarcoma, occur, but not frequently, in the extradural fatty tissue. Teratoma in this situation are equally rare.

Intradural Tumors.—Intradural tumors are still less frequently of a metastatic nature. Fibromata, sarcomata, endotheliomata, and myxomata are all found and neuromata may occur on the nerve roots. Inclusion cyst of the dermoid variety is a rare condition, of which I have seen one example, and of which Hale White has reported a typical case (*Transactions of the Clinical Society of London*, 1900, p. 140). It is frequently, though not necessarily, associated with developmental defects in the cord itself, as Harrichausen pointed out (*Deutsches Zeitschrift für Nervenheilkunde*, 1909, Band xxxvi, Heft 3 and 4, p. 268). It usually affects the lower part of the cord and the region of the cauda equina. Circumscribed tuberculomata may affect the spinal pia-arachnoid and cysticerci may also develop in the arachnoid space. Echinococcus cyst may develop intradurally, although they are far more common extradurally.

Such intradural tumors may spring from the inner surface of the dura, from the arachnoid tissue, or from the pia. In the latter case they frequently involve the cord substance itself, although the pia mater does offer some protection. These tumors are generally single, with the exception perhaps of sarcomata and neuromata, which are often multiple. Melanoid sarcoma frequently has multiple metastatic growths affecting the pia, both of the brain and cord. Usually these intradural tumors owing to the limited space in which they develop are small and generally spherical in shape, or if larger they develop in the longitudinal direction and have a cylindrical appearance. They lie usually to one side or in some cases posteriorly and less frequently on the anterior surface of the cord, between this and the bone, in such a way that the cord becomes compressed. Those in the region of the cauda equina may reach a moderately large size.

Meningeal Tumors.—Meningeal tumors may, it is evident, cause symptoms in three directions: (1) by involvement of the bone, (2) by compression of the nerve roots, and (3) by pressure on the cord. Intradural tumors will certainly be more likely to act in the last two directions first, while in extradural tumors there is the probability of its extending longitudinally between the membrane and the bony covering for some distance, without causing anything more than the symptoms of involvement of the nerve roots, the dura acting as a very efficient protection to the cord. Of course as the tumor extends laterally so as to partially surround the cord, pressure symptoms will be produced, but the course of least resistance for its development will be longitudinal.

In some cases the nerve roots may be severely compressed and the cord may be quite indented, and yet be able to recover its functions if the pressure be relieved. In other cases compression myelitis

sets in early with softening and irreparable destruction, due to the occlusion of the blood supply to the part. With hard and rapidly growing tumors compression myelitis occurs more quickly, as the vessels have no chance to adapt themselves to the new conditions. Pial tumors may not only compress the cord but may in their growth break through the pia and infiltrate the cord substance itself.

Intramedullary Tumors.—Tumors of the spinal cord itself—intramedullary tumors—are in the majority of cases of a gliomatous nature. Sarcomata, endotheliomata, and also tubercle are also found, usually having invaded the cord from the pia. Generally these intramedullary tumors are primary and solitary. They may be circumscribed or infiltrating. Among the former we find sarcomata and tubercle; usually surrounding them there is a small area of softening. Gliomata are practically always of an infiltrating nature, spreading chiefly longitudinally, often extending throughout the whole length of the cord and even into the medulla; they are often associated with cavity formation and give rise to the condition known as syringomyelia, which is discussed under a separate heading. In rare and less typical cases it forms a circumscribed firm tumor mass giving rise to the signs of compression of the cord and the nerve roots. In meningeal tumor the symptoms of root compression usually precede those of compression of the cord, while in the intramedullary tumors the latter appear first, giving rise to some minor peculiarities in the symptomatology, which will be discussed later. Intramedullary tumors are not, as a rule, very extensive, save, as in gliomata, in the long axis of the cord, although they may cause considerable increase in size in the cross-section as well.

Symptoms.—The symptoms produced by cord tumors, whether extra- or intramedullary, are in general very similar; they are those of a more or less slow compression of the cord and the nerve roots, as is seen also in vertebral tumors when they begin to involve these structures. The points of differential diagnosis have been referred to already. The first signs are almost always those of root irritation, and usually the sensory roots suffer first, giving rise to pain. The location of these pains will naturally depend on the site of the tumor; they are usually, in the early stages, confined to one side, but may later become bilateral. In rare cases when the tumor is situated on the dorsal surface of the cord the pains may be bilateral from the beginning as in vertebral tumors. They are of extreme severity and obstinacy, although sometimes in the early stages when the tumor affects the dorsal cord there may be free intervals while the tumor, having destroyed one nerve root, is extending to involve the next. The pains are usually of a neuralgic, lancinating character, confined to the area of distribution of the affected roots. In meningeal tumors the signs of involvement of the motor roots appear after those due to involvement of sensory roots, both in time and intensity. The motor nerve involvement is evidenced by cramps in individual muscles with paresis and atrophy and disturbances in the electrical reactions. In the early stages these,

too, are usually unilateral. Owing to the overlapping of the various nerve roots in their distribution to the skin, the involvement of a single one never causes any appreciable loss of sensibility. In the same way a single motor root can be destroyed without a complete paralysis of any individual muscles. With a tumor lying intradurally where it may involve many nerve roots before causing actual cord compression, we may get corresponding areas of loss of sensibility and atrophic paralysis of the muscles supplied by the affected roots, with the characteristic changes in their electrical excitability.

When the cord itself is affected there results widespread paralysis, both motor and sensory. If the tumor be laterally situated we get the characteristic symptoms of a Brown-Séquard paralysis with motor paralysis of a spastic nature below the level of the lesion on the same side due to the involvement of the pyramidal tracts, and also we will find a loss of sense of position in the muscles, more or less marked; in this paralyzed limb, due to the posterior columns being affected. On the opposite side below the level of the lesion there will be found an impairment or loss of sensibility to pain, heat, and cold, due to the involvement of those fibers in the anterolateral tract after they have crossed over. At the level of the lesion there may be signs of involvement of the motor and sensory nerve roots with the atrophic paralysis of the muscles supplied and the loss of sensibility in the distribution of the nerve roots affected. Naturally this will be seen on the side of the tumor. Later as the tumor progresses the other side is involved and we get a complete paraplegia below the site of the lesion. With pressure on the nerve roots, the sensory roots first show the evidences of the compression; but with pressure on the cord, the motor involvement is as a rule more noticeable in the early stages. With the complete paraplegia there is usually retention of urine and obstinate constipation. Decubitus forms over the sacrum, purulent cystitis and pyelitis by extension follow, then paralysis of the bladder, and death is caused by some such complication.

If the implication of the cord goes on to a complete and total lesion, and this may come on gradually and slowly as the result of increasing pressure, or rapidly and suddenly as the result of local edema or softening of the cord due to the vascular involvement, the picture changes. The muscles which before were spastic now become flaccid and the tendon-jerks, formerly exaggerated, now disappear. This is not peculiar to tumors but follows any complete section of the cord.

It is only necessary to take up now in some detail the differentiation of tumors involving the lower segments of the cord and the conus terminalis and those involving the corresponding nerve roots in the cauda equina. In the case of a tumor of the conus the development of the symptoms is more rapid and there is comparatively little pain of an irritative sensory root nature, while in the case of a tumor involving the cauda the onset is very slow and associated with intense pain radiating along the course of the sciatic nerves and in the region of the bladder. Loss of sensibility in the affected parts appears early in

the course of tumor of the conus, and is often in the form of a dissociated anesthesia, while in a caudal tumor it occurs late in the disease and is then usually complete.

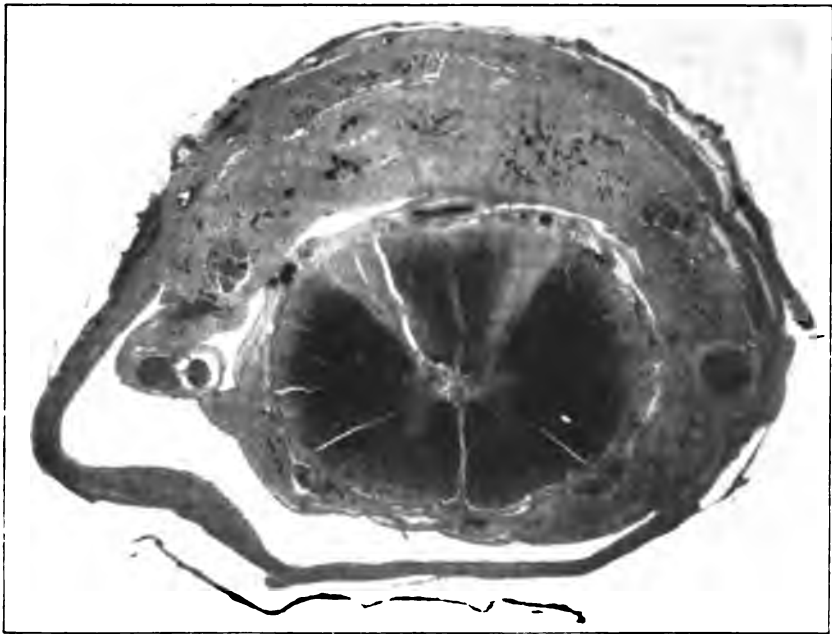
Symptoms of motor paralysis originate early and atrophy with the reaction of degeneration rapidly follows in a tumor of the conus, while in the caudal lesion motor paralysis comes on more slowly, and atrophy and electrical changes may not appear for a still longer time. In tumors of the conus the motor paralysis preponderates, while in those of the cauda the symptoms of the irritation of the sensory roots are the most striking. In the former, again, the bladder and rectum are paralyzed early, while in the latter this disturbance may be practically absent until the very latest stages.

Diagnosis.—The diagnosis of a tumor of the spinal cord is then based not only on the grouping of the symptoms but on their successive character. In the typical case we see first the gradual involvement of a nerve root on one side, then with the further growth the cord is compressed first on one side and later bilaterally. Pachymeningitis cervicalis hypertrophica may give rise to symptoms which might easily be mistaken for a tumor growth, but it always affects both sides of the cord simultaneously and very extensively, giving rise to a widespread bilateral atrophic muscular paralysis of the arms, with a correspondingly widespread impairment of sensibility, due to the extensive nerve root involvement, before there are any signs of actual cord compression. (Plate XLVI.)

Having made a diagnosis of tumor of the cord it is essential to localize its level in the cord, in other words, what segments are involved. By referring to the tables showing both the motor and sensory segmental distribution, the upper level of the lesion can be localized with certainty. It is then necessary to study the relation of the segments and nerve roots to the spines of the vertebræ, at their exit from the cord in order to be able to say with accuracy just where the tumor is.

Treatment.—In tumors of a syphilitic nature only is medication of any value. Surgical interference is the only hope in all other forms. The success of such treatment will vary considerably with the site of the tumor—as has been already said vertebral tumors do not offer much hope for permanent good results. Intramedullary growths, from their situation, cannot be removed without causing too great a destruction of the cord tissue; meningeal tumors, however, and these are the most common, hold out a good prospect of successful removal, and, when surgical interference has not been postponed too long, of complete recovery. As has been mentioned before, surgical interference is often indicated in cases, in order to relieve by section of the sensory roots, the excruciating pain due to their irritation, even when there is no chance of attempting to remove the tumor growth. Pressure on the cord having been diagnosticated, when efficient surgical aid can be obtained, it is as much the duty of the physician to advise surgical intervention as it would be if he diagnosticated obstruction of the bowel.

PLATE XLVI



Pachymeningitis Hypertrophica Cervicalis.

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SPINAL CARIES

Spinal caries, or Pott's disease, was first described by Percival Pott in 1779, who called attention not only to the spinal disease but to the cord symptoms as well, believing them to be due to the direct pressure on the cord by the displaced bones. Since then great advances have been made not only in the study of the disease in the vertebræ but also in the pathogenesis of the cord affection and at the same time in the treatment of the disease.

Etiology.—Tuberculosis of the spine is almost always secondary to a tuberculosis in other organs, most frequently in the lungs, but occasionally in the lymph glands. In a few cases, especially in adult life, it is apparently the primary focus. Injury to the spinal column frequently appears to be of some importance as an indirect agent, probably by creating a focus of lowered resistance. In such cases there is usually a history of a more or less long interval between the time of injury and the onset of the symptoms, but one realizes that the disease in the bone is often very slow in developing. It is possible that the disease may be already present in the bone in some cases so that a comparatively slight injury will produce a breaking down of the diseased vertebræ with signs of pressure on the cord. In the majority of cases spinal caries develops without any apparent exciting cause.

The disease may affect individuals of any age. Gowers states that he has found it more frequent in childhood after the age of three, and next in early adult life, and this has been my own experience. Finkler, however, found that 50 per cent. of his 20 cases were over fifty years of age. Vertebral tumors, carcinoma, and osteosarcoma, and syphilitic disease of the vertebræ, may also cause a breaking down of the bodies and gibbus formation with compression of the cord, and these are considered under their separate headings.

Pathological Anatomy.—The disease begins usually in the body of the vertebra, less frequently in the intravertebral disks or joints, and still more rarely in the laminæ. One vertebra alone may be affected or several may be involved. According to Van Rey's statistics from 114 cases the lumbar region was more frequently affected and especially the third lumbar vertebra. The middle dorsal region is the next in frequency and the cervical region still less frequently involved. Compression of the cord follows disease of the dorsal vertebræ more often than when the cervical or lumbar regions are affected.

The disease begins as a tuberculous osteomyelitis or periostitis of the vertebral body, or more rarely in the intervertebral disks and may spread with more or less rapidity to the neighboring vertebræ or may remain confined to one. With the progress of the disease there is a breaking down of the affected bone, it may be involving several vertebræ, so that owing to the body weight they gradually collapse, and as the arches are seldom affected, there is the slow formation of an angular deformity or "gibbus" of the spine, with possibly pressure on the cord with the kinking of the canal.

Compression of the cord is especially likely to occur if this gibbus formation occurs suddenly as a result of injury with breaking up and dislocation of the diseased bone. It may occur also as a result of an extension of the tuberculous process into the extradural fatty tissue; or again it may result from the formation of an abscess on the inner surface of the vertebræ between them and the cord. The most common cause of compression of the cord is the extension of the tuberculous process into the epidural tissue; it follows when the periosteum of the vertebræ has become affected, and the long posterior ligament destroyed, so that the bacilli have direct access to the loose fatty tissue. Granulation tissue rich in vessels forms at the site of invasion with, very often, numerous small tuberculous nodules. The fatty tissue disappears and the vessel walls show a tuberculous endarteritis which may go on to thrombosis. Caseation results sooner or later depending on the richness of the blood supply. It appears as a rule first in that part next the vertebral bodies while on the dural surface, and the upper and lower extremity of the mass, fresh tuberculous nodules may still be present. The dura, as a rule, forms a very efficient protection against the further invasion of the bacillus, and while its superficial layers may become affected it is very rarely penetrated. The growth may become quite extensive extradurally, both in its long diameter, especially when several vertebræ are affected, and also in thickness so as to cause compression of the cord.

Compression occurs, too, as a result of abscess formation. It may happen that an abscess in the vertebral body breaks through the periosteum and the long posterior ligament and disrupts into the spinal canal. It may later be absorbed, at least partially, or become caseated in part, and the remnant of it may stay for years, if the patient has not already succumbed to the complications of a paraplegia. The slow formation of an angular curvature may occur without any disturbance of the cord. The more rapid the formation of the gibbus the more likely is there to result signs of cord compression.

The dura reacts in the same manner as does the pleura in the face of a tuberculous lesion. It becomes thickened by the formation of new layers of fibrous tissue on its inner or medullary surface with the formation also of new vessels. Hyaline changes are usually found in the vessel walls and frequently there is found numerous psammoma bodies. Adhesions form between the dura and the pia-arachnoid matting them together into one. Schmaus describes a tuberculous endarteritis in such cases and attributes the cord changes to a localized edema from obstruction in the veins or lymph passages. In this way the variations in the paraplegia, and ultimate complete recovery in many cases after a long period, are accounted for. In some cases, owing to compression, a condition of ischemia results with consequent softening of the cord.

The changes in the cord itself are chiefly in the white substance; the axis-cylinders are swollen and the myelin sheaths dilated and thinned. Or in more advanced cases the nerve fibers and cells may be broken

down and degenerated and then numerous spaces are left in the neuroglia when the axis-cylinders have disappeared, large granular cells may be found in recent acute cases. In older chronic ones one gets more or less well-marked ascending and descending degeneration secondary to the lesion.

Descending degeneration will be found below the site of the lesion, wherever it may happen to be, in the crossed pyramidal tract as far as the upper sacral segments, and in the direct pyramidal tract to the beginning of the lumbar enlargement. And also in the ventral part of the posterior columns, a small tract which may be followed a short distance below the site of the injury. Degeneration of certain descending paths in the anteromedian region of the cord also occur.

Ascending degeneration above the site of the lesion will also be found in the posterior columns, in the direct cerebellar tracts, and both ascending and descending fibers will be found in the anterolateral or Gowers' tracts.

Symptomatology.—The first symptom pointing to the vertebral disease is usually a dull pain in the affected part, which is exaggerated by movement, especially by stooping over. It may not always be present, and especially in children is apt to be absent even in the presence of extensive vertebral disease. In other cases the pain is not definitely localized but appears to be general in the spinal column. There is usually associated with this pain more or less rigidity of the back which may often be noticed when the patient raises himself in the bed or attempts to lie down again. Localized tenderness on pressure over the spinous processes may very often be elicited, although in some cases it fails.

In other cases the nerve roots are involved early and the first symptoms are those of root pain, due to irritation of the sensory nerves. These pains are often of a very severe stabbing character in the area of distribution of the affected roots; usually they are bilateral and it is not uncommon to get herpetic eruption following such irritation of the posterior ganglia. Paresthesiæ in the affected extremities are commonly among the early symptoms complained of, a feeling of numbness or frequently of formication; there may also be a subjective feeling of coldness, though objectively the parts feel normally warm.

These root symptoms may be entirely absent in some cases, even when the vertebral disease is quite widespread, and the first symptoms may be those of compression of the cord. There then follows disturbances in the innervation of the voluntary muscles, at first merely a feeling of heaviness in the legs; they tire more easily and the movements are slower and more uncertain. The rapidity of the progress of the disease varies considerably, but sooner or later, if neglected, the weakness of the legs becomes more marked until a definite paraplegia develops with increase in tone, increased knee-jerks, and all the signs of a lesion of the pyramidal tracts, with disturbances of function of both bladder and bowels. Spasmodic twitchings and cramps in the legs may be very troublesome. The motor paralysis is, as a

rule, more evident, especially in the early stages, than is the sensory impairment. This may vary considerably and almost any degree of impairment may be found below the level of the lesion. Owing to the retention of urine cystitis develops readily and pyelitis is apt to follow; bed-sores, too, have to be guarded against, and death may follow from pyemia or some intercurrent complication, or from the lighting up of the primary focus in the lungs or elsewhere.

Under proper conditions, however, improvement and even recovery may take place even after the evidences of cord involvement are well marked. The improvement in the symptoms will be observed to follow inversely the order of their appearance—disturbances of sensation usually clear up first and later the paralysis. The improvement may of course come to a halt at any stage, depending on the severity of the cord involvement. The most necessary requisite for a permanent improvement, is the cure of the disease in the vertebræ, without such, any improvement can of course be only temporary.

The symptoms will vary somewhat according to the site of the lesion. If the dorsal cord be affected, as is most commonly the case, there will be paralysis from the waist down, with spasticity and increased tendon-jerks, and the plantar response will be of the extensor type. The abdominal and epigastric reflexes will be lost below the level supplied by the affected segments of the cord, but may be obtained above this. There may be difficulty in starting micturition, or indeed complete retention of urine with incontinence of overflow. The more rapid the onset of the paraplegia the more likely is there to be this involvement of the sphincters. The bowels are usually obstinately constipated. In mild cases there may be little or no impairment of sensation below the level of the lesion.

When the cervical enlargement is affected there will be involvement of the arms as well; they will show as atrophic flaccid paralysis due, as a rule, to pressure on the nerve roots, and a loss of sensation will be found on their inner surfaces corresponding to the nerve roots involved. On the trunk the sensory loss when present will extend to the second costal cartilage. The lower extremities will be in a condition of spastic paraplegia.

When the upper cervical region is affected the neck muscles will be paralyzed, and the patient will have difficulty in raising his head. The diaphragm and intercostals may all be paralyzed and the patient may have to depend on the accessory muscles for respiration. The arms and legs will then be in a state of spastic paralysis. When the lesion is high up the hypoglossal nerve and the muscular branch of the accessory are often affected. Retropharyngeal abscess may sometimes be made out in such cases. Disease affecting the lumbar enlargement will give rise to a flaccid atrophic paralysis of the lower extremities with loss of reflexes and incontinence of urine and feces.

Diagnosis.—When the spinal deformity is well developed, before the onset of the cord symptoms, there is, as a rule, no difficulty in the diagnosis. In rare cases the cord symptoms appear first, and in these

the diagnosis may be obscure for some time. Repeated examination of the spine will usually clear up the doubt before long by finding the vertebral tenderness and slight irregularity of the spinous processes. When the disease affects the cervical cord it may simulate pachymeningitis cervicalis hypertrophica, but will be differentiated by the presence of the disease in the bone and in the less extensive involvement of the nerve roots. Amyotrophic lateral sclerosis will be distinguished by the bony tenderness and by the sensory impairment. The *x*-rays will often assist materially in clearing up the diagnosis in these cases.

In children disease of the spine is practically always tuberculous, but in elderly people metastatic malignant tumors sometimes affect the vertebræ. In these cases there will be the evidence of the primary growth to throw light on the condition. According to Gowers the root pains in malignant disease of the vertebræ reach a degree of severity seldom seen in caries, but this is only a relative distinction and in my experience the pains are often very severe in caries.

Prognosis.—Varies with the age of the patient; among other things, being much more favorable in young people than in those advanced in life. Even when paraplegia is complete a marked improvement and even recovery is by no means uncommon under suitable conditions. The site of the disease will also influence the prognosis, it being worse when the lumbar region is involved with paralysis of bladder and bowels, and the strong tendency to trophic disturbances than when the lesion is above this level. When the disease involves the upper cervical cord and the centres for the diaphragm the danger, of course, is materially increased. Apart from these points one may be guided to some extent also by the degree of sensory impairment present. When this is slight it shows that the involvement of the cord has not reached a severe grade, but even when the sensory impairment is complete recovery is not always out of the question. When the paralysis of the legs is of the spastic type and the tendon jerks are increased, the prognosis is better than when the paralysis has become flaccid and the reflexes have disappeared. The signs of tubercular disease elsewhere must be taken into account in giving a prognosis, and the powers of resistance of the individual to the infection should be calculated.

Treatment.—The first essential in the treatment of these cases is rest to the diseased parts. We must strive primarily to cure the diseased vertebræ, for without this being accomplished any improvement will be merely temporary. Immobility of the affected spine is the first object to be aimed at. In young people and children this can be best accomplished by a plaster mould fitted to the back and extending from the head to the upper part of the buttocks. This should be lined with absorbent cotton to render it perfectly comfortable. The child can be held in this by straps, and in this way can be carried about, always in the recumbent position, and moved into the open air whenever advisable. The necessities of nature can be attended to without any soiling of the bedclothes, or if such occurs, cleanliness, so necessary to prevent bed-sores, can be obtained without disturbing the back.

Graduated pressure can be exerted on the spinal kyphosis, when this is advisable, by filling in the depression in the plaster mould.

In older patients rest in the recumbent position is just as necessary and may be combined with some form of extension. When the cervical spine is affected, extension may be applied by a weight hung over the head of the bed on a pulley and attached by a strap to the patient's head; the head of the bed may be raised so that the weight of the body acts as a counterextension. In lumbar caries the weight may be attached to the feet and the foot of the bed raised similarly. In dorsal disease extension may be applied both to the head and the feet.

The second requisite is to build up the general health of the patient, first by means of a full nutritious diet, including plenty of eggs and milk; cod-liver oil has for a long time enjoyed a well-earned reputation in such cases; iron tonics of various kinds may also be indicated; and secondly, by keeping the patient in the open air and sunlight as much as possible. Counterirritation to the spine opposite the diseased region, once so commonly used, is no longer considered either indicated or useful. After a prolonged rest in the recumbent posture, if the vertebral disease seems quiescent a plaster jacket may be applied in such a way as to support the body weight on the crests of the iliac bones, and also to retain the immobility of the spine. Or if the cervical spine has been affected some sort of jury-mast or support under the chin and occiput should be arranged to carry the weight of the head, and the patient may then gradually be allowed up.

Should there be a tendency to contractures in the muscles of the extremities, massage and passive movements properly carried out will prevent these. Forcible passive movements should not be resorted to while the vertebral disease is still active, at least without great care being exercised to prevent any disturbance of the spine. Tenotomy is seldom necessary to overcome the contractures but may be resorted to in suitable cases. In some cases laminectomy and removal of diseased tissue has been of service but should never be resorted to before an attempt has been made to bring relief by rest. It would, however, be advisable, when rest has been given a thorough trial and proved unsuccessful, or in cases where the compression of the cord is becoming steadily worse under the rest treatment, and should there be danger of a permanent paraplegia setting in, unless, of course, there was severe or advancing tuberculosis in other organs.

CAISSON DISEASE

Caisson disease or diver's palsy is a disease that occurs in men who have been exposed to great atmospheric pressure when they come too quickly under normal atmospheric conditions. It is usually seen in divers or men working in caissons at some depth, usually a depth of 25 to 30 meters under the water, where they are exposed to an atmospheric pressure of three to four times the normal. The symptoms never come on while the individual is exposed to the greater pressure

but only when he comes under the normal atmospheric conditions too rapidly after such exposure. Rapid decompression is the chief cause of the accident; hunger and exhaustion appear to be accessory etiological factors in some cases.

Symptoms.—The symptoms may be cerebral or spinal—and their severity depends to some extent on the degree of variation in the pressure to which the individual has been exposed. They are more apt to occur in individuals who are new to the work than in those who have become accustomed to it.

The cerebral symptoms are, as a rule, slight and transient; they consist of a bursting feeling in the head, dizziness, ringing in the ears, and nausea. The spinal symptoms may be more serious. Usually there is a period of from five to fifteen minutes after the diver leaves the water before any symptoms appear. In some cases paralysis is preceded by loss of consciousness, of variable duration; in other cases vertigo, severe pains in the limbs and back and tingling were observed before paralysis developed.

Paraplegia, temporary or permanent, is the commonest form of paralysis. In severe cases there is often at the onset paralysis of the arms as well, but this may rapidly disappear, leaving a permanent paraplegia with sensory impairment and bladder disturbances. The paralysis of the lesser depths, that is, from 25 to 30 meters, is usually transitory, but in men working at the greater depths, that is from 40 to 65 meters, the paralysis is often permanent and not infrequently death occurs either suddenly on their exit from the water, or coma sets in and death follows in some hours. Boinet believes hemorrhages into the cord to be the cause of the serious symptoms of this disease and claims to have verified this by autopsies on human beings and by experiments. The lesions affect the whole of the spinal system with the exception of the anterior horns and do not pass above the level of the second dorsal segment. Hemorrhages into the spinal meninges occur also. Zografidi described actual small aneurysmal dilatations of the vessels of the cord as well. It is believed that the hemorrhages into the spinal cord are produced in two ways: (1) the bubble of gas liberated from the blood by too rapid decompression, actually causes rupture of the capillaries in the substance of the cord; (2) hemorrhagic infarction following occlusion of a vessel of the cord by bubbles of gas is thought to occur. According to Paul Bert nitrogen is dissolved in the blood in increased quantity at high pressure, according to Dalton's law, and is liberated in the form of bubbles during decompression.

Treatment.—The air embolism theory finds support from the good results of slow decompression and from the therapeutic action of recompression followed by slow decompression in cases of illness. Slowness of decompression is the true prophylaxis. In caissons the time given for coming out should be at least three minutes per atmosphere. In the case of divers this is scarcely possible, but the use of more rigid diving suits and allowing at least one minute per atmosphere for decompression would prevent most accidents due to compressed air,

CHAPTER XIV

DISEASES OF THE OPTIC THALAMUS, MIDBRAIN, AND CEREBELLUM, AND THEIR TREATMENT

By GORDON HOLMES, M.D., M.R.C.P.

OPTIC THALAMUS

THOSE large masses of gray matter which constitute the basal ganglia of the brain have received little attention from clinicians, and until the publication of Dejerine and Roussy's paper in 1903, and the further elaboration of the subject by Roussy in his monograph of 1907, little or nothing was known of the symptoms which result from disease of the optic thalamus.

Anatomists had long recognized that the thalamus is a large relay station in the course of the sensory path, as all fibers which pass cerebralward from the brain-stem terminate in it and there transfer the impulses they carry, probably as Monakow suggests through intercalated cells (*Schaltzellen*), to the thalamocortical neurones; but clinical observers had on the whole attached little importance to destructive lesions of the thalamus as a cause of sensory disturbance. Charcot, for instance, insisted that the hemianesthesia often found with lesions in its neighborhood was due only to involvement of the posterior limb of the internal capsule, and even Nothnagel in 1885 held that there were not sufficient facts to connect any form of sensory disturbances with thalamic lesions. Similarly, there was much dispute in the early days of modern neurology as to whether lesions of the thalamus produce paralysis, and though Broadbent, Meynert, and Gowers pointed out the association of hemiathetosis, hemichorea, and other involuntary movements with lesions of it, any causal relationship between them was vigorously denied by others, especially by Charcot and his school. Reflex, vasomotor, and sphincter disturbances have been also described as symptoms of thalamic lesions, but not with sufficient support to receive general recognition.

It is almost wholly to Dejerine and his pupils that we owe the knowledge of the clinical symptoms of thalamic disease that we now possess, and especially to Roussy, who defined the "syndrome thalamique" with remarkable completeness and accuracy in his thesis, and supported his clinical observations and deductions from experiments on animals.

Thalamic Syndrome.—According to Roussy the "syndrome thalamique" is characterized by hemianesthesia, and especially loss of deep sensibility, with persistent, paroxysmal spontaneous pains in

the affected side; there is little or no hemiplegia, and no rigidity or contracture; hemiataxia is usually present, and frequently also irregular involuntary movements of the choreic or athetoid type in the affected limbs.

A considerable number of cases with these clinical symptoms had been already described, several prior to the appearance of Roussy's monograph, and the postmortem examinations which have been possible in some of them have justified this clinical definition and have confirmed his statement that these symptoms result only from lesions which involve the posterior and external part of the thalamus.

These are the essential and the most prominent and characteristic signs of thalamic disease, but the relative prominence of the individual symptoms may vary, and any one or other of them may be absent in certain cases.

From a recent review of 10 cases in which the lesion was confirmed by autopsy, it was found (Head and Holmes) that involuntary movements were present in only 4, hemiataxia in 7, and spontaneous pains in 8. Similarly, though some degree of sensory loss existed in all, its intensity varied from only slight disturbance of one or other form of sensibility to practically complete loss of all components of sensation.

Further, other symptoms may or may not occur according as the lesion remains limited to the optic thalamus or extends into neighboring parts. Hemiparesis, if it exists, is due, not to disease of the thalamus itself, but to involvement of the internal capsule which lies on its outer side, and it depends on the extent to which this is affected. It is a noteworthy feature that even when it is relatively severe it is, as a rule, not associated with any rigidity, and that the plantar reflex on the affected side may not be of the extensor type.

Hemianopsia, which is observed in some of the cases, naturally occurs when the lesion spreads into the pulvinar of the thalamus or to the external geniculate body; and the partial deafness which is rarer is probably due to extension of the disease toward the median geniculate body or its peduncle.

In the great majority of the cases which have been hitherto observed the lesion has been of vascular origin, either a hemorrhage or thrombosis; I have seen one case in which a small and apparently localized hemorrhage occurred after a blow to the head. A few cases have been also observed in which the condition was apparently due to a tumor involving the thalamus.

A further discussion of these usual symptoms of thalamic disease would be out of place here, but reference must be made to the nature and significance of certain of them which are more or less characteristic, and for which patients afflicted by the thalamic syndrome would probably come under treatment; these are the involuntary movements and hemiataxia, and the sensory disturbances with spontaneous pains or painful paresthesiæ.

It must be recognized in the first place that in the overwhelming majority of the cases the lesion is stationary and non-progressive, and

as it is usually the scar of a thrombotic or hemorrhagic softening it cannot be regarded as a persistent irritant stimulus, while the symptoms it produces may persist unaltered for years or even increase gradually in intensity. Another explanation must be therefore sought for these symptoms that indicate overactivity of some nervous mechanism, and especially for the involuntary movements and the spontaneous pains.

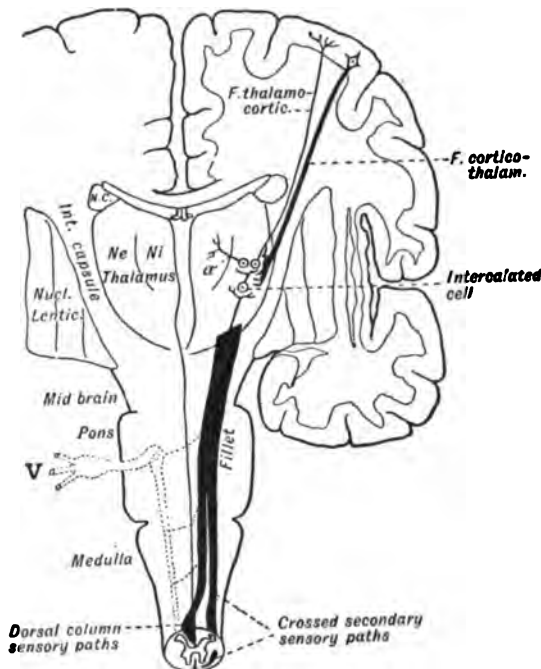
Hemiataxia.—The hemiataxia which is so common a symptom is certainly a defect phenomenon due, in the majority of the cases, to involvement of the cerebellothalamic neurones of the superior cerebellar peduncle which have an important function in the regulation of voluntary movement. In certain cases, however, it may be a result of the loss of sensation, and especially of loss of the sense of position, and of the appreciation of movement.

Involuntary and Spontaneous Movements.—These on the other hand are so obviously a symptom of overactivity that they cannot be a *direct* result of a destructive lesion; for Hughlings Jackson's axiom, that negative or destructive lesions cannot produce directly positive symptoms or symptoms of overactivity, must be accepted. Their occurrence is now generally attributed to the fact that the destructive lesion of certain centres frees other nervous mechanisms from the control which normally regulates their activity. It must be to this uncontrolled activity of such centres that the spontaneous movements are due. Which are the inhibitory or controlling centres that are destroyed in these cases, and which are the other centres to whose overactivity these motor symptoms are directly due is too complicated a matter to receive discussion here. Two facts may be, however, emphasized; first, that the actual region of the thalamus which must be involved to produce these movements, is that through which the cerebellothalamic or rubrothalamic fibers pass, or in which they terminate; this is in the ventral and caudal regions of the nucleus. Secondly, it is necessary that the pyramidal tracts should not be severely injured; the spontaneous movements are always absent if there is severe paralysis, and they usually set in only as the paresis which supervenes as an acute lesion is passing off, and, as I have observed in more than one case, they cease immediately on the onset of another attack of hemiplegia, or with any considerable progress of the weakness. An excellent instance is afforded by Horsley's case in which violent athetosis of an arm was permanently checked by removal of the corresponding cortical motor centres. It may be therefore concluded that the involuntary movements which occur in the thalamic syndrome are due to the removal of the inhibitory activity of a cerebello-rubro-thalamic mechanism on certain motor systems, and one of these at least is the motor cerebral cortex.

Loss of Sensation.—The loss of sensation which is the most constant of the symptoms of thalamic disease is evidently due to injury of some part of the sensory path which passes through and undergoes a relay in the thalamus. But evidently there may be important differences in the form of the loss of sensation according as the sensory path is

involved before its thalamic relay, that is in the fillet, or after, that is in the thalamocortical neurones. But as might be expected from its anatomical arrangement, in gross lesions of the thalamus the sensory path is usually injured in both places, though the symptoms are generally predominantly those of a supra- or infrathalamic (nuclear) interference.

FIG. 56



To show the position and relations of the optic thalamus in the central sensory path. Two distinct paths exist in the spinal cord; a crossed secondary path in the ventrolateral column which conveys impressions of pain, temperature, and touch, and a second uncrossed path in the dorsal column which also carries touch, and in which run impulses that underlie the sense of position, the appreciation of movement, the discrimination of two points, and the recognition of vibration, size, shape, form, weight, and consistence. This second path decussates in the lower part of the medulla oblongata, but runs separate from the first path at least as high as the pons. All these secondary sensory fibers, now crossed, terminate in the ventrolateral region of the optic thalamus. The impressions they carry are regrouped here and, through intercalated neurones, are distributed along two distinct paths; the one carries impressions to the cerebral cortex, the other, we assume, toward the more mesial parts of the optic thalamus. The cortico-thalamic fibers, which terminate in the lateral nucleus of the optic thalamus, are also shown. (Head and Holmes.)

Pains of Central Origin.—The occurrence of pains of central origin, that is, such as are not apparently due to any peripheral stimulation, has excited much discussion; but to understand their nature and treatment it is necessary to consider another sensory phenomenon that seems to be constantly associated with them. This is the tendency for stimuli of the unpleasant order to produce more pain and discomfort on the affected side than in normal parts, despite the diminution of sensibility that usually exists. Pain, whether produced by prick or pressure,

scraping with the finger nails or any sharp object, and extreme degrees of temperature (*i. e.*, below 15° C. and above 50° C.), all evoke a stronger reaction and give rise to more pain and discomfort than on the normal side of the body. But still more remarkable is the fact that a parallel overaction to pleasurable stimuli may be frequently obtained from the affected regions; mild degrees of warmth, for instance, may be more pleasant than on the normal side, and tickling, erotic, and other stimuli may excite a much stronger emotion from it.

Thus there is associated with these spontaneous pains of the thalamic syndrome an excessive response to affective stimuli, though there may be a diminution of all forms of sensibility on the affected side. And this seems to be the most constant and most characteristic feature of thalamic disease. The most reasonable explanation of it that can be offered is one analogous to that given of the involuntary movements. For as this sensory disturbance persists unaltered for years; as it is generally associated with stationary and non-irritant lesions; and as it occurs almost constantly with lesions in this region of the sensory tract, but rarely with lesions elsewhere, it cannot be regarded as a phenomenon of irritation. If, on the other hand, it were looked upon as due to the removal by the lesion of the inhibitory control of one centre upon another which is normally concerned with the affective components of sensibility, this explanation would offer an exact and reasonable interpretation of all the associated phenomena.

Numerous investigations have made it very improbable that the afferent components of sensibility reach any portion of the cerebral cortex; their centre must be consequently situated subcortically. Further, the subcortical mass of gray matter which is most intimately associated with the sensory path, and which offers the best opportunities for this function is the optic thalamus. And other arguments tend to prove that it is here we must seek the centre for the affective elements of sensibility.

It must be remembered that Roussy originally pointed out, and his observations have been confirmed, that these lesions which produce spontaneous pains and the associated overaction to affective stimuli lie in the lateral region of the thalamus where they must interrupt the fibers connecting the cortex with the thalamus, as these pass from the internal capsule into the latter. And if we assume that by so doing they remove a control which the cerebral cortex, the organ of discriminative sensibility, exerts on the thalamus, the centre of the affective elements of sensation, there would result a condition of overactivity of the latter which must show itself, first by an exaggerated reaction to these elements; and secondly by the occurrence of affective sensation under conditions which are not recognized as naturally capable of producing it, and these sensations would consequently appear to be spontaneous.

By such an explanation, which is merely outlined here, the most characteristic symptoms of thalamic disease, the spontaneous or involuntary movements, and the spontaneous or central pains with the

associated overreaction to affective stimuli, would be due to the same cause—they would result from a disturbance of the inhibitory control which one part of the nervous system normally exerts on another.

FIG. 57



To show the position of lesion which is outlined, in a typical case of the thalamic syndrome. *a*, ext. med. lam.; *b*, int. med. lam.; *c*, ant. nucl.; *d*, tenia thalami; *e*, thalamic radiations; *f*, V. d'Azay; *g*, pes; *h*, fornix.

Treatment.—The treatment of cases of thalamic disease is difficult and unsatisfactory; the most common lesions are of vascular origin, and we can do nothing to repair the destruction they cause. Neoplasms are rarer and most serious as they cannot be accessible to surgical intervention. Tuberculous tumors, however, are not uncommon in the thalamus and may become latent, but gummas which generally grow from the cerebral membranes are naturally rare.

I have observed cases of slow origin and progressive course in which the symptoms, either unilateral or bilateral, have suggested disease of the thalamus. These have occurred in relatively young subjects and seemed to be due to degenerative lesions, though I have had no opportunity of confirming the diagnosis or determining the nature of the disease; one of them at least seemed to improve under large doses of iodides.

The treatment of diseases of the optic thalamus must be consequently mainly symptomatic. The special symptoms for which these patients

seek relief are the hemiataxia which interferes with purposive movements of the limbs; the violent and troublesome involuntary movements, and the severe persistent paroxysmal pains which practically characterize the condition.

For the hemiataxia we can unfortunately do nothing; no care in attempting reëducation seems to have the slightest effect, and in addition to the ataxia purposeful actions are usually further disturbed by uncontrollable spontaneous movements. When slight these require no treatment, as they are not seriously troublesome to the patient, but they are occasionally so violent and troublesome that relief becomes urgently necessary. Only surgical measures can afford it. Two methods have been tried. In one case Horsley removed the cortical motor centres of the opposite hemisphere of the brain; this paralyzed the affected arm almost completely and abolished the movements which had been previously so violent that even at this cost of paralysis the relief was extremely grateful.

Spiller devised another method for the treatment of severe athetosis, which was adopted with apparently some success in one case at least. He observed that the flexors of the arm were more severely involved than the extensors, and so it occurred to him that if some of this excessive innervation of the flexors could be, so to speak, switched into the extensors by nerve transplantation, a more nearly normal relation between these groups of muscles and their opponents might be established, and thus the athetoid movements would be lessened. In the case referred to he recommended lateral anastomosis of the divided ulnar and median nerves into the musculospiral nerve, and in a second operation the circumflex and musculocutaneous nerves were divided and an end-to-end anastomosis effected between the central end of one and the peripheral end of the other.

In slight cases there is no doubt a certain amount of relief may be obtained by keeping the patient as quiet as possible, as all physical and mental excitement exaggerates the movements.

But the spontaneous pains are the symptoms which most urgently need relief. Roussy pointed out, and Edinger, Grief, and others had observed previously, that a feature of these pains is that they cannot be controlled by ordinary analgesics. This is undoubtedly true, though a certain amount of alleviation may be sometimes obtained by phenacetin, antipyrine, and especially aspirin, but large and frequent doses are unhappily needed. Anyone of these drugs may be advantageously combined with sodium bromide, especially in cases where sleep is poor or disturbed. I have given the following prescription with some success:

R—Sodium bromide	gr. xij or xv
Phenacetin	gr. viij or x
Tincture of gelsemium	℥vii
Water	℥ss

Three or four times daily.

In other cases opium or morphine may be necessary, and though there may be a natural reluctance to exposing the patient to the danger of acquiring the morphine habit, it must be remembered that the suffering is in many cases very intense, and that without relief life is scarcely worth living. I have seen good results from giving urethane in 20 or 30 grain doses two or three times a day, during a period when the pains were severe and sleep was disturbed.

Apart from medicinal treatment much may be done for these central pains. I have repeatedly observed that they are more severe, or more probably that the patient is less tolerant to them, when his health is low, and that he seems to suffer less when stronger and in better condition. All possible measures of improving the general health should be therefore adopted.

Still more important is the avoidance of all external conditions that may excite or increase the pain; all stimuli containing affective elements may do this, and of these the most common is cold. Any exposure to a draught, washing in cold water, and above all a change in temperature may excite or aggravate the pain, and the patient should be consequently warned to expose himself to them as little as possible. As a rule it will be found that the patients have to clothe themselves more than adequately. I know several patients who winter and summer wear a warm glove on the affected hand, as "the hand cannot bear the cold."

As has been shown (Head and Holmes) any emotional stress and excitement may induce attacks of pain or painful paresthesiæ, and it is consequently advisable to save the patient from worry or excitement.

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DISEASES OF THE MIDBRAIN

As the midbrain is the sole pathway between the brain and the lower levels of the nervous system, and as it also contains many important special centres, any disease or injury of it may produce numerous symptoms, some of which are characteristic, while others, as a spastic motor hemiparesis, may result from lesions in any part of the long tracts which pass through it. In order to deal concisely with the interpretation of these symptoms for diagnostic purposes it will be most convenient to consider separately those that result from affections from each of its three divisions, namely, the crura, the tegmentum, and the corpora quadrigemina, though clinical disease can be rarely limited accurately to any one of them.

Syndrome of the Crura Cerebri.—By ordinary definition the crura cerebri are understood to consist of the long tracts that run longitudinally on the ventral surface of the midbrain and which more anteriorly are continuous with part of the internal capsule. These are the corticopontine bundles, and the corticospinal or pyramidal tracts. This part of the brain-stem is injured most frequently by hemorrhages or softenings, or by tumors growing either within the midbrain, or from the skull or meninges and compressing it. Gummata are not infrequent in this region.

The characteristic symptoms of such lesions constitute the syndrome of Weber, or a spastic paresis of the opposite side of the body including the face with palsy, either partial or complete, of the homolateral oculomotor nerve, owing to injury of it as it passes outward through the crus. The intensity of the hemiplegia as well as the extent of the oculomotor palsy depends on the size and the site of the lesion, but the ophthalmoplegia tends to be more complete than when the lesion affects the oculomotor nucleus, since the root fibers collect together as they reach the interpeduncular space. These are the only signs of a lesion limited to the peduncle; sensory disturbances are absent, and other cranial nerves are not involved.

It must be remembered that the coexistence of a hemiplegia with palsy of the opposite oculomotor nerve may be due to two or more separate lesions, and that this is especially common in cerebrospinal syphilis. The history of the simultaneous onset of the two palsies is therefore important.

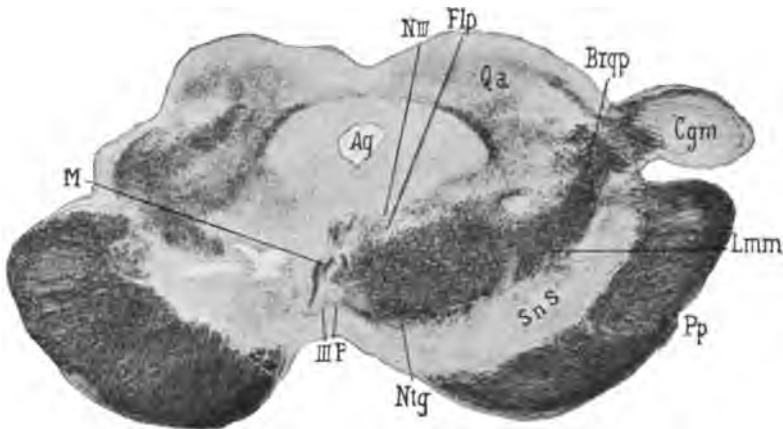
Lesions in the Tegmentum.—More numerous and more variable symptoms are produced by disease of the tegmentum owing to the several centres and paths that are contained within or pass through it. It is most commonly involved by tumors, especially tuberculomata and gliomata, but hemorrhage and softening frequently occur in it. As Martial has shown it is not infrequently the site of the lesion in post-traumatic hemiplegia, especially when the injury was in the neighborhood of the orbit.

Some degree of hemiplegia or paresis of the opposite side may exist, but only when the crus is involved or compressed by the lesion. Sensory disturbances on the contralateral half of the body are almost constant, but as the secondary sensory trigeminal neurones—quinto-thalamic fibers—run, in part at least, separate from the main sensory tracts, sensibility may be either more or less affected on the face than on the trunk and limbs. The intensity of this sensory loss depends on the site and the extent of the lesion; all forms of sensibility are usually disturbed, and probably in no other part of the nervous system is sensation so commonly affected in all its elements as in the midbrain.

Hemiataxia.—More important localizing symptoms, however, are the hemiataxia and unilateral spontaneous movements so frequently associated with disease in this region. The hemiataxia has been attributed to the sensory disturbances that usually coexist, but though these may

contribute to it its nature in my experience almost invariably indicates that it is of cerebellar origin and is due to a lesion of the superior cerebellar peduncles—cerebello-rubro-thalamic fibers, their terminal centres

FIG. 58



A softening in the left side of the tegmentum of the midbrain involving the mesial fillet, *Lmm*; the nucleus ruber, *Nig*; the oculomotor nerve and its nucleus, *iii*; the dorsal longitudinal bundle, *FLP*; and part of the substantia nigra, *SnS*. (Halban and Infeld.)

FIG. 59

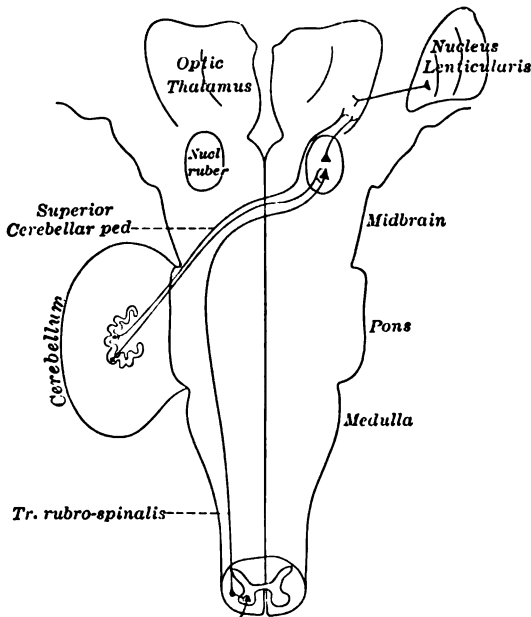


Diagram to illustrate the centres and paths, lesions of which produce tremor and other forms of involuntary movement.

in the nucleus ruber or in the posterior end of the optic thalamus, or of the efferent rubrospinal tract. The distribution of the ataxia also confirms this opinion. The superior cerebellar peduncles run direct from their origin through the posterior part of the tectum and tegmentum of the midbrain, then decussate in the commissure of Wernekinck to terminate in the opposite red nucleus and optic thalamus; consequently we might expect to find the hemiataxia homolateral to the lesion and on the side opposite to the hemiparesis and sensory loss when the lesion is situated posteriorly; bilateral when the commissure is involved, and on the same side as the motor and sensory paralysis when the disease lies anterior to this level. And these are the facts we do find. Frequently, as Ferrier and Turner showed by experiments on animals, the ataxia resembles intention-tremor when the superior peduncles are injured.

Tremor.—But more characteristic as a topographical sign of disease in this region is the presence of spontaneous or involuntary movements which generally take the form of tremor. This fact was first pointed out by Benedikt, and the coexistence of unilateral tremor with paralysis of the opposite third nerve has become known as the syndrome of Benedikt. The tremor often resembles very closely that of paralysis agitans; it is relatively regular in rate, range, and amplitude, and of a frequency of 3 to 5 oscillations per second. It occurs as a rule only when the limb is wholly or partially unsupported. It is not under volitional control, and it generally becomes a well-marked intention-tremor on movement. Occasionally the spontaneous movements are more irregular or choreiform, and even athetosis may occur, but this is more characteristically associated with lesions of the optic thalamus than with those of the midbrain.

The exact pathogenesis of these spontaneous movements has been much discussed. The original view favored by Charcot and others attributed them to a partial or irritative lesion of the pyramidal tract, but numerous clinical facts disprove this hypothesis; it is indeed now recognized that in order that they may occur it is essential that the pyramidal tract should not be severely injured. On the other hand many facts tend to prove that tremor may result from a destructive lesion of any part of the superior cerebellar peduncle, or of the nucleus ruber in which it terminates, or of the rubrospinal tract or v. Monakow's bundle which passes from this to the spinal cord. It therefore seems to depend on a lesion of the cerebello-rubro-spinal system when the pyramidal tracts are relatively intact (Bonhoeffer, Holmes). If this be so the tremor must be homolateral to the lesion if the cerebellar peduncle is affected before its decussation, contralateral if the lesion lies above the decussation or involves the nucleus ruber, and again homolateral if it affects the rubrospinal tract after its crossing. And this we actually find; since in the majority of cases it depends on involvement of the nucleus ruber, it is generally found on the same side as the sensory and motor disturbances and on the side opposite to the lesion.

PLATE XLVII

Fig. 1



Paralysis of Upward Movement of the Eyes, showing the excessive wrinkling of the forehead in the attempt to look up.

Fig. 2



Tumor of the Pineal Body, showing the effect of pressure on the mid-brain and the hydrocephalus of the lateral ventricles that it has produced. (Bailey and Jelliffe.)

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This tremor is generally associated with hypertonicity of the muscles and rigidity of the limbs, and this increases the danger of confusion of these cases with paralysis agitans. Sometimes, indeed, the face too is rigid and mask-like.

Ophthalmoplegia.—The most common focal sign of midbrain lesions, however, is the presence of ophthalmoplegia due to injury of the whole or some part of the oculomotor or trochlear root or nucleus, or possibly of a supranuclear ocular centre. The most common condition is a partial or complete paralysis of the third nerve on the side of the lesion. The intrinsic ocular muscles, however, frequently escape in lesions of the tegmentum, though isolated palsy of them is, in my experience, not uncommon with disease in the anterior end of the midbrain or in the optic thalamus. This must be due to the fact that their centre is situated the most anteriorly in the oculomotor nucleus. Paralysis of the superior oblique muscle, due to involvement of the trochlear nerve, may occur when the disease extends to the posterior part of the midbrain. (Plate XLVII, Fig. 1.)

Less common though more characteristic are the palsies of the conjugate or associated ocular movements; much the most frequent of these is palsy of the upward movements of both eyes, but downward movement may be affected either alone or together with this, or the power of associated convergence of the eyes may be lost. Different explanations have been offered as to the causes of these conjugate palsies; some, as Tödter, have assumed the existence of a supranuclear centre for these conjugate movements in the tegmentum of the midbrain or in the corpora quadrigemina; others, as Spiller, attribute them to direct injury of the oculomotor nuclei; and finally von Monakow would explain them by the interruption of the fibers connecting the two oculomotor nuclei. There are probably not sufficient facts available to decide the question definitely; though the existence of a supranuclear conjugate centre would explain their occurrence most easily, it must be admitted that there is no other direct evidence of it. Conjugate palsies of lateral movements of the eyes may occur also with midbrain lesions, owing to interruption of the fibers of the dorsal longitudinal bundle connecting the sixth with the oculomotor nucleus, though they are more characteristically associated with pontine disease in the neighborhood of the abducens nucleus.

Pseudobulbar Palsy.—Another symptom of disease of the tegmentum of the midbrain which is not uncommon is some degree of pseudobulbar palsy; it is probably due to the fact that the pyramidal fibers destined for the innervation of the motor cranial nerves leave the corticospinal tracts in the midbrain and pass downward through the tegmentum near to the raphe. When these are interrupted the facial muscles become spastic and weak, any expression the face assumes tends to become fixed, and there may be tendency to excessive emotional display; the tongue also becomes weak and spastic, swallowing may be difficult, and speech indistinct and monotonous, or there may be even considerable dysarthria.

Symptoms of Disease of the Quadrigeminal Bodies.—Disease in this region produces fewer focal symptoms and is less easy to diagnosticate. Tumors are the most common pathological agents; they generally grow within the brain, but may, as those that spring from the pineal body, only compress it.

Tumors of the roof of the midbrain usually produce a more or less characteristic syndrome. Headache and vomiting are constant and are often very severe, and optic neuritis occurs in a large proportion of the cases. The other symptoms are drowsiness which often sets in early, an irregular staggering and reeling gait, though the individual movements of the lower limbs may not be incoördinate, and either irregular ocular palsies or paralysis of the conjugate vertical movements, associated with nystagmus. The pupillary reactions are less commonly disturbed. Hearing may be affected when the disease involves one posterior quadrigeminal body, though this is probably not a prominent symptom unless it extends to the median geniculate body; hearing is then generally reduced in both ears, but more so in the ear of the opposite side. As the anterior quadrigeminal body is not a true visual centre in man disturbances of vision do not occur unless the disease extends into the pulvinar thalami or to the lateral geniculate body.

Tumors of Pineal Body.—Tumors of the pineal body, with the symptoms of which we have become acquainted by Marburg and the compilations of Bailey, Jelliffe, and Kidd produce the general symptoms of cerebral tumor with certain focal signs, and there may be in addition characteristic disturbances of nutrition. The general symptoms, those of increase of intracranial pressure are, as a rule, well marked, as hydrocephalus almost always coexists, for owing to its position the tumor may block the aqueduct of Sylvius and thus obstruct the flow of cerebrospinal fluid; it is also liable to compress the veins of Galen and thus impede the venous return. Headache, vomiting, optic neuritis, and vertigo therefore occur and may be associated with other symptoms of hydrocephalus. The focal signs are those of disease of the corpora quadrigemina, namely, irregular or associated ocular palsies, pupillary disturbances, staggering gait, ataxia, and occasionally cerebellar asynergia and dysdiadokinesis. The metabolic symptoms are unnatural adiposity, sexual precocity, with premature development of the primary and secondary sexual characters, and in some cases cachexia. As the pineal undergoes involution after about the seventh year these constitutional symptoms may be expected only in young subjects. (Plate XLVII, Fig. 2.)

The exact relation of the adiposis to the pineal disease is questionable; it may be a direct result, or it may be due to atrophy or functional disturbance in the hypophysis owing to its compression as a result of hydrocephalus. The sexual precocity probably depends, as Marburg suggested, on the nature of the tumor; teratomata or adenomata which produce an increase of the secreting tissue of the gland would be most liable to produce it.

Treatment of Midbrain Disorders.—This must depend on the nature and site of the lesions to which they are due. We are unable to repair the damages produced by vascular lesions and can rarely do anything to compensate the symptoms that result from them. Tumors which lie within or involve the midbrain cannot be accessible to surgical intervention, and few of those that compress it can be safely removed. It may be, however, in the future possible to operate with success on certain tumors of the pineal gland, as we have now learned to diagnose them with fair certainty. It must be remembered that tuberculous tumors, which are especially common in this region, may become latent and their symptoms may diminish or even disappear. Gummata, which may respond to antisyphilitic treatment, are also not uncommon at the base of the midbrain.

Few of the individual symptoms, too, are amenable to treatment or require separate consideration. The ataxia must be treated on the lines laid down in the chapter on the cerebellum. Tremor or other spontaneous movements are rarely so severe from disease of the midbrain as to require radical treatment, but if so violent as to be a source of discomfort they may be dealt with as suggested in the chapter on the diseases of the optic thalamus.

The ocular palsies are even more unsatisfactory from the point of view of treatment; unless due to compression of the nerve or its nucleus by a gumma that will yield to treatment we can do nothing to remove their cause. Ptosis, it must be remembered, is often an advantage as it obviates the discomfort of diplopia. When the ocular palsies have resulted from a non-progressive lesion, as a softening, it may be advisable to consider the treatment of the squint by operation, as this may at least remove the deformity, and it may improve vision considerably.

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THE INTERPRETATION AND TREATMENT OF CEREBELLAR DISORDERS

An acquaintance with the anatomical connections and the functions of the cerebellum is necessary in order to be able to interpret the symptoms of its diseases and devise the proper and most adequate methods of treatment. But the cerebellum is the head-piece or centre of an

extensive system of centres and tracts the functions of which are closely related to its own, while their disturbance may produce symptoms very similar to those of cerebellar disease. It is therefore advisable to include in the term "cerebellar disorders" not merely those conditions which are directly due to disease of the cerebellum itself, but also to deal with the symptoms which arise from disease of the related systems. A short review of the anatomy of these centres and systems, and a brief *resume* of their functions is consequently essential for the purpose of this chapter.

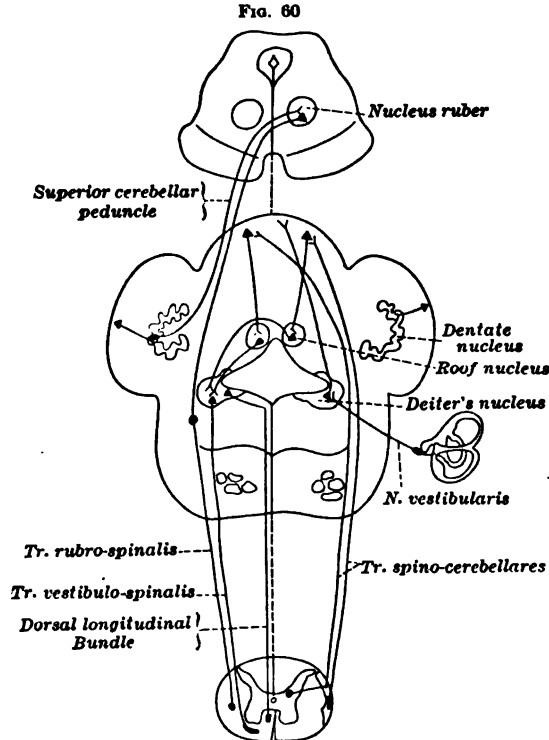


Diagram to illustrate the spinal connections of the cerebellum. On the right the afferent tracts are represented, and the efferent cerebellar tracts on the left.

Anatomy of Cerebellum.—The cerebellum, that large unpaired organ which is constantly present throughout the vertebrate kingdom, has connections with all parts of the central nervous system, from the lowest part of the spinal cord to the cerebral cortex. These connections take place through its three paired peduncles. The inferior peduncle or corpus restiforme is mainly afferent, the middle peduncle is wholly so, while the superior cerebellar peduncles, or the brachia conjunctiva, are wholly efferent, though one important tract, the tractus spino-cerebellaris ventralis, or Gowers' bundle, enters the cerebellum in close relation to them.

Through these peduncles the cerebellum is intimately connected

in both the afferent and efferent directions with almost all other parts of the central nervous system. Those parts which are especially differentiated in relation to the cerebellum and correlated in activity with it may be spoken of as the cerebellar systems.

These cerebellar systems may be briefly sketched as arc mechanisms with afferent and efferent limbs, while their essential and controlling centres lie in the cerebellum.

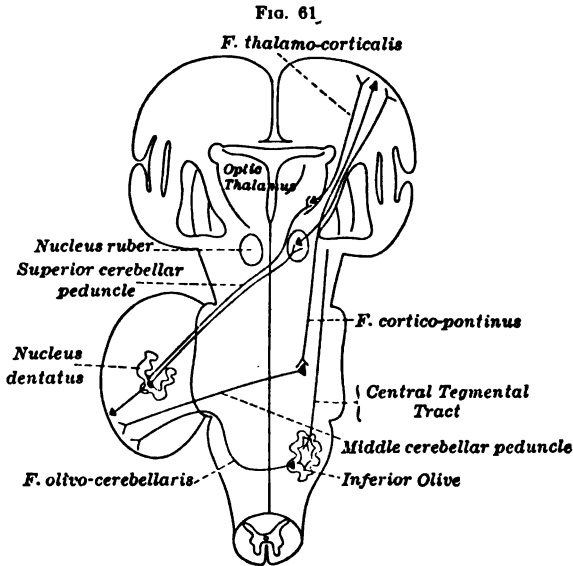


Diagram to illustrate the afferent and efferent connections of the cerebellum with the forebrain.

The spinocerebellar arc consists of the afferent spinocerebellar tracts, or the direct cerebellar tract of Flechsig and Gowers' bundle, which terminate in the cerebellar cortex; by means of projection fibers this is connected with the central nuclei, and from these two efferent paths conduct to the cord. The one takes origin in the dentate nucleus, passes to the opposite red nucleus, and from this the rubrospinal path after decussation leads to the cord. The other path starts in the roof nuclei and passes through the semicircular fibers of the cerebellum to Deiters' nucleus, and thence through the vestibulospinal and the dorsal longitudinal bundles to the cord. It must be noted that the cerebello-fugal connections of the cerebellum are homolateral with the spinal cord, *i. e.*, the connection of the cerebellum in this direction is direct, not crossed, while on the afferent side the relation is also at least predominantly homolateral. A consequence of this is that it may be expected that the symptoms of unilateral cerebellar disease should show themselves predominantly on the side of the lesion.

On the other hand the connection of the cerebellum with the forebrain and basal ganglia is crossed in contralateral. On the afferent side we have each cerebral hemisphere connected by the corticopontine

tract, the nuclei pontis and the middle cerebellar peduncle with the opposite lateral lobe of the cerebellum. The basal ganglia are probably similarly connected with the cerebellum by the central tegmental tracts which pass direct to the inferior olives, and by the crossed olivocerebellar fibers that take origin from these.

Similarly the efferent cerebellocerebral connections are crossed, owing to the decussation of the superior cerebellar peduncles. These terminate directly in the optic thalamus, and through this they can undoubtedly, by the thalamocortical fibers, act on the cerebral cortex.

Finally the cerebellum receives another important system of fibers from the primary vestibular nuclei, which enter it by passing on the inner or mesial side of the corpus restiforme. These vestibulocerebellar fibers form the inner portion of the peduncle, or the corpus juxta restiforme. The vestibular nerve, which carries impulses from the semicircular canals, terminates chiefly in the dorsal vestibular nucleus, but also sends fibers to Deiters' nucleus and the nucleus of Bechterew that lies immediately dorsal to it; and numerous fibers take origin in these primary vestibular nuclei, chiefly from Deiters' nucleus, and pass to the cortex of the vermis, probably on their way giving collaterals to the roof nucleus of the same side. Though described by Edinger it seems doubtful if any vestibular root fibers pass directly to the cerebellum.

The Functions of the Cerebellum and its Related Parts.—Probably no part of the central nervous system has been the subject of so many investigations, experimental and physiological, clinical and pathological, as the cerebellum. It is impossible to deal with the results of these investigations here, but a short reference to the more important is advisable owing to the aid it will be in interpreting the significance of the clinical symptoms to be dealt with in this chapter.

Its extensive connections with all other parts of the central nervous system, and the intimate afferent and efferent relations in which it stands to them, would suggest, *a priori*, that the cerebellum is in some way concerned with the functions of all these parts and that its duty may be to coördinate their activities. Again viewed from its phylogenetic history it may seem that its development corresponds closely, as Edinger has pointed out, to the relative complexity or variety of the motor activities of the animal. It is small and rudimentary in the mud fishes, larger and more complex in those which swim freely; it is simple and relatively small in the reptiles which crawl or glide on the earth, relatively enormous in the birds which fly through the air. And even in many classes of mammals a similar relation is noticeable, though the development of the lateral lobes parallel with that of the forebrain tends to obscure it.

The physiological investigations that have thrown most light on the functions of the cerebellum have been extirpation experiments; the results of stimulation though valuable as complementary, have been less important.

Seventy years ago Flourens described the cerebellum as a special organ for the coördination and regulation of voluntary movements and discovered that lesions of it disturbed the harmony and interrelationship of muscular contractions. Though this view has been criticised, in whole or part, by subsequent investigations it expresses an opinion which has come to be generally held. Schiff was impressed by the tremor and unsteadiness of animals from whom part of the cerebellum was removed, and offered the explanation that this is due to a disturbance of innervation which upsets the normal relation of muscles to their antagonists and synergics. Ferrier has regarded the cerebellum as a coördinating centre and one specially concerned with the regulations of the muscular activities necessary for the maintenance of equilibrium; while Munk, too, attributes to it the function of maintaining equilibrium.

Rolando suggested that the cerebellum may be a source or reservoir of nervous energy, and Luys and Weir Mitchell emphasized feebleness as the most prominent persisting symptom of destructive lesions of the cerebellum.

But the investigations of Luciani are the best known and the most complete. He describes, as a result of removal of one-half of the cerebellum, a series of symptoms limited to or more marked on the same side of the body, which supervene as the irritative effects of the operation pass off. When analyzed these symptoms are seen to be due to *atonía* or defective tone in the muscles; *astasia*, or tremor and unsteadiness which shows itself in irregularity of movement and tremor or oscillations of unsupported parts of the body owing apparently to incomplete or irregular fusion of the individual (or fundamental) muscular contractions; and finally *asthenia*, or feebleness of the affected muscles.

But these views, too, have failed to find universal acceptance. The *astasia*, or the tremulousness in movement and in the maintenance of attitudes is generally admitted, but a deficiency of tone is denied by Ferrier and others, though in my opinion wrongly. Thomas, too, disputes the statement that hypotonia occurs as he has not observed it in patients afflicted with chronic cerebellar disease. Paresis and feebleness of the affected muscles as a direct result of cerebellar lesions is even more generally disputed, and there is no doubt that even in acute cerebellar lesions it is rarely prominent, though it certainly occurs. Others, as Ferrier and Munk, regard the maintenance of equilibrium as the chief function of the cerebellum. According to Ferrier, by disease or injury of it equilibration is disturbed owing to disarrangement of the muscular synergics that are necessary to correct the displacement of the centre of gravity that must occur in every step. Similarly, Munk ascribes the disturbance of equilibration to an upset of the combined or general movements (*Gemeinschaftbewegungen*) of the vertebral column and limbs.

Thomas, too, emphasizes the importance of the cerebellum as a reflex centre for equilibration, and for the maintenance of the body

in the erect attitude at rest or during reflex, automatic or voluntary movement.

These views which ascribe to the cerebellum as its chief function the association and coördination of the muscular activities necessary for equilibrium raise the interesting point of the relation of the labyrinthine and cerebellar functions. Their intimate anatomical connection suggests that their functions must be closely related. Removal of a labyrinth, or section of one vestibular nerve, produces symptoms in many points similar to those of acute destruction of one side of the cerebellum; equilibrium is disturbed and the homolateral limbs become hypotonic, unsteady, and remain for a time even feeble. On the other hand the ataxia of cerebellar disease is absent, and any disturbance of movements is due chiefly to the hypotonicity. Thomas suggests that while the vestibular apparatus is concerned chiefly in the maintenance of the head and trunk in passive attitudes, the cerebellum controls equilibrium in active movement.

Further, orientation in space is ascribed to labyrinthine activity, and though this may not be a direct function of the labyrinth, it seems to depend largely on the state of tonicity of the muscles which is under the regulating control of the labyrinthine impulses. The cerebellum, on the other hand, seems to take no part in it.

Though the cerebellum receives so many afferent paths, and though it has actually developed in the afferent bulbar system, it cannot be regarded as a sensory organ; there is no clinical evidence, and it is only in man that conclusive observations can be made, that any of the impulses that reach it or pass through it ever affect consciousness directly or indirectly as a sensation, or that they can underlie any form of conscious sensation. It must be, however, mentioned that Risien Russell described anesthesia and analgesia of the same extent as the motor affections after destructive lesions of one and both sides of the cerebellum. Magendie originally supposed the cerebellum to be the organ of "muscle sensibility." This idea has been revived by Lewandowsky, who regards cerebellar ataxia as a "sensory ataxia" due to disturbance of muscular sensibility. Munk also adopts the suggestion that the cerebellum is concerned with muscular sensibility, but this opinion has not been confirmed by any other physiologists, and from numerous observations I can deny positively that any form of sensation is affected in man by either acute or chronic cerebellar lesions. Lotmar described defective appreciation of weight in limbs affected by cerebellar ataxia, but as others I have failed to find any confirmation of this opinion in the examination of several cases of cerebellar disease.

Finally, Sherrington's genial definition may be referred to. According to him the cerebellum is "the head ganglion of the proprioceptive system," that is of the non-sensory components of the large afferent system that collects its impulses from the end-organs which are affected by changes in the body itself—from the muscles, tendons, and joints, as well as from the labyrinth. This extensive proprioceptive system, which also sends centralward the impulses that underlie the appre-

ciation of position and movement, *i. e.*, those which constitute what is commonly spoken of as "the muscle sense," has, as Sherrington says, its own reflexes which are mainly concerned in the maintenance of muscle tone, and in preserving the attitude of the body and regulating the contractions and relaxations of the muscles on which this depends. These are the peripheral impulses which furnish the cerebellum with the materials that subserve its functions.

What is the functional relation of the cerebellum to the cerebrum? Anatomically there is a very close connection by double crossed paths, but on the physiological side it is not yet possible to define their interrelationship in any short formula. Russell, however, and later Luciani, have shown that after extirpation of one side of the cerebellum the motor centres in the contralateral cerebral hemisphere are more excitable than normal.

If we attempt to summarize in general terms the conclusions in regard to the functions of the cerebellum to which physiological experimentation has led, we may say that it is an organ which by its tonic action controls the activities of those portions of the nervous system that are concerned in the initiation or production of movement, so that each movement is continuous and maintained and is executed with the greatest possible preciseness in range and direction; and that by the elaborate coördination of the various effective centres over which it presides the groups of muscles which coöperate in producing movements, or in maintaining the attitudes necessary for their execution, work harmoniously.

Finally we can conclude that though it has developed in the afferent system and has extensive afferent connections *it is not a sensory organ*; nor, on the other hand, can it be regarded as motor, though it sends efferent tracts directly or through relays to other positions of the nervous system.

A short reference may be also made to the specific functions of the different afferent and efferent connections of the cerebellum as revealed by their experimental destruction, though our knowledge of them is very incomplete and indefinite. Marburg and Bing have each divided the spinocerebellar tracts and described more or less identical symptoms; the chief were hypotonicity of the homolateral limbs and an awkwardness and ataxia of gait which seemed to be due chiefly to incoördination of the "Prinzipalsteegungen" of the limbs, or of those muscles regulating the relation of the limbs to the shoulder girdle and pelvis. Thus they resemble the symptoms produced by a unilateral cerebellar lesion. But these symptoms disappear rapidly, even after bilateral section. The importance of the afferent impulses carried by these tracts for the coördinating functions of the cerebellum cannot be doubted, and the relatively slight and transient effects their section produces probably indicates that apart from them there is another spinal path open to cerebellopetal impulses. The fact that section of the inferior cerebellar peduncles, which probably includes these other paths (*e. g.*, spino-olivo-cerebellar) produces more intense and persistent

cerebellar symptoms, may be taken as an argument in favor of this view. The nature of the afferent cerebellar components of vestibular origin has been already referred to. It is probably on these that the tonic and equilibrating functions of the cerebellum mainly depend.

Significance of Cerebro-ponto-cerebellar System.—The significance of the cerebro-ponto-cerebellar system is obscure. We know practically nothing of the effects of disease or section of the cerebro-pontine fibers; this is due to the fact that with them other cortico-efferent tracts (*e. g.*, corticospinal) are generally involved, and the pure effects of the disturbance of the cerebro-cerebellar connections are therefore obscured. Mills has suggested that as Türck's bundle takes its origin chiefly from the region of cortical representation of the labyrinth it may be intimately concerned with equilibration; while Kleist believes that perseveration of voluntary muscular contraction, and a stiffness of the opposite limbs may result from disease of the frontopontine tract.

Function of Cerebello-thalamo-cortical System.—Of the exact functions of the efferent cerebello-thalamo-cortical system we are also ignorant; we may recall, however, the hyperexcitability of the opposite cerebral hemisphere after extirpation of one side of the cerebellum described by Luciani and Russell, and perhaps deduce from it that by this system the cerebellum exerts an inhibiting or controlling influence on cerebral motor activity. The cerebello-rubro-spinal and cerebello-vestibulo-spinal efferent paths are probably more important, but of their exact significance we know very little. There are many facts, however, which indicate that Deiters' nucleus, which is the chief relay station in the cerebello-vestibulo-spinal system, is largely concerned with the control of muscle tone, and its intimate relation to the vestibulum suggests it. Thiele, too, has shown that the decerebrate rigidity of Sherrington persists on repeated section of the brain-stem down to the level of Deiters' nucleus, but disappears when this is injured.

Function of Cerebello-rubro-spinal System.—The cerebello-rubro-spinal system may be more intimately concerned with the determination of muscular contractions and their proper fusion. Ferrier has pointed out that tremor and an irregularity of movement very similar to the intention-tremor of disseminated sclerosis results from section of the superior cerebellar peduncles, and in the chapter on Diseases of the Midbrain we have seen that lesions of the nucleus ruber and even of the rubrospinal tract may produce the same effects.

Symptoms of Cerebellar Disease.—We can come now to the symptoms of cerebellar disease, or the disturbances of function that result from lesions of the cerebellum. The remarks we have already made have prepared us for the consideration of these symptoms and their interpretation. But we must emphasize at once that the combination of symptoms depend largely on the form of the disease, and especially on its evolution. Lesions of sudden or rapid onset, as softenings, hemorrhages, rapidly growing abscesses and tumors, or the effects of extensive surgical interference, produce symptoms which resemble closely those observed after experimental lesions of the cerebellum or

its related systems. But if the lesion is non-progressive or becomes stationary these symptoms diminish and some of them may eventually disappear. On the other hand, diseases of slow evolution, as the primary atrophies and degenerations of the cerebellum and slowly growing tumors, evoke, as a rule, less prominent disturbances. Finally, it is now well known that the not uncommon congenital lesions of the cerebellum may fail to show any clinical evidence of their existence.

It is advisable, however, to point out first the clinical symptoms of cerebellar disorders, and later to consider their relation to the type of the disease.

Ataxia.—Ataxia, or incoördination of movement, is almost invariably the most prominent sign. If contrasted with the ataxia of tabes dorsalis, which is chiefly due to loss of those components of sensation by which movement is controlled, *i. e.*, especially loss of those conscious afferents from the muscles, tendons, and joints that subserve the appreciation of posture and movement, it is at once seen that it differs therefrom by the fact that the sense of position and the appreciation of movement in the ataxic limbs are unaffected. The tabetic can partly compensate this sensory loss by voluntarily controlling his movements by vision, and his ataxia is consequently greater when his eyes are closed or he is otherwise unable to guide his movements by his eyes; in cerebellar disease, on the other hand, as the irregularity of movement is due to a disturbance of a central coördinating mechanism which the patient cannot overcome either by the aid of sensation or vision, its manifestations are equally great whether his eyes are closed or open. The ataxia of cerebellar disease is a primary incoördination due to a defective coöperation of the various muscles, agonists, antagonists, and synergics, concerned in the act. It must be realized that few if any movements of our bodies and limbs result from isolated contraction of a single muscle, for, as Sherrington especially has shown, even the simplest reflex contraction of a muscle is accompanied by a relaxation of the tone of its antagonist; and it is evident that under other conditions the uniformity and regularity of a movement may be regulated by the reciprocal innervation of the antagonists of the contracting muscle. Further, even the simplest acts require for their completion the contraction of synergic muscles to prevent accessory movements at the joint concerned, for example that of the extensors of the wrist when the fingers are flexed in order to prevent flexion of the hand. Finally, to execute any purposive act it is essential that the more proximal segments of the limbs should be fixed in an appropriate attitude by the contraction of its muscles. Consequently even a simple act, as grasping a pen, necessitates the harmonious coöperation of many groups of muscles, *viz.*, the flexors of the fingers as agonists, the relaxation of their antagonists, the extensors of the fingers, the concentration of the synergic extensors of the wrist to prevent the flexion of the hand by the contraction of the flexors of the fingers, and finally the tonic contraction of the fixators of the elbow and shoulder. It is this harmonious coöperation which is disturbed by cerebellar lesions.

Consequently cerebellar disease produces a disproportion in the elements of a movement, and there results therefrom what Babinski has termed a *dysmetria*. If a patient so affected is asked to bring his forefinger to the tip of his nose the hand does not follow the direct and natural line of movement but deviates from it, and is then voluntarily drawn back to the correct direction; it is also frequently interrupted in the line of movement and the finger may finally pass the spot it desires to reach. Thomas rightly points out that spontaneous move-

FIG. 62



ments are executed slowly and in stages instead of uniformly, so that an irregularity resembling that seen in disseminated sclerosis may result. This dysmetria is well illustrated (Thomas) by a patient attempting to take a glass of water in his affected hand: in approaching the glass the hand is opened unduely widely, then it is brought so abruptly to the glass as to spill its contents, and finally the object may be grasped with a disproportionate or ill-measured force.

As may be expected from this analysis of cerebellar ataxia, it is always most prominent in "movements d'ensemble," that is, large movements which require the coöperation of numerous groups of muscles, while simple movements may be well executed. This is another point in the distinction of cerebellar incoördination from that of *tabes dorsalis*.

Another component of cerebellar incoördination has been emphasized by Babinski under the term *asynergia*, or a loss of the faculty of associating movements. If a normal person is requested to place one foot on a chair in front of him he does so by simultaneously flexing his hip and knee; a subject of cerebellar ataxia will, however, probably first raise his leg by flexing his thigh, and only later flex his knee: the two acts are not simultaneously executed. Similarly, though the patient may have difficulty in maintaining his equilibrium as he walks he does not aid himself by swinging his arms in association with the movements of his legs.

Another test of *asynergia*, which well illustrates its effect on gait, is to ask the patient to throw his head backward; in attempting this he may fall owing to failure to readjust his balance by the simultaneous flexion of his knees. To this *asynergia* the difficulty in equilibration is largely due: movements which displace the centre

of gravity, as every step must do, do not excite reflexly the normally associated muscular contractions, which again throw the centre of gravity over the base; in other words, the body does not adjust itself immediately to its altered position.

Equilibrium.—We have seen that many physiologists have regarded the inability to maintain equilibrium in the erect posture as one of the most prominent signs of cerebellar lesions in animals, and a difficulty in equilibration is unquestionably often the most pronounced symptom of cerebellar disease in man. Whether this is due to a specific disturbance of equilibration as occurs in labyrinthine disease, or is merely the result of the ataxia and its consequent dysmetria of movement and asynergia is difficult to decide, but my own opinion inclines to attribute it predominantly to the latter causes. It may be observed, in severe cases at least, not only when the patient stands but even as he sits unsupported; then the body sways from side to side and the attempts at balancing by his arms and head are awkward and ineffectual.

The ataxia which results from unilateral cerebellar lesions shows itself predominantly, and often almost exclusively, in the homolateral limbs; occasionally, however, the ataxia is almost bilateral, but this is chiefly the case with tumors which often have a diffuse or widely indirect effect. It is usually more marked in the leg, especially in the movements of locomotion, than in the arm. The face and the tongue are rarely affected, but, as we shall see later, phonation and articulation are often disturbed. When unilateral the ataxia of the trunk cannot be demonstrated with certainty, though the asynergia between the trunk and the lower limbs is certainly an important factor in the disturbance of gait. The dissimilarity of the instability in gait and in standing in *tabes dorsalis* and in cerebellar disease is further shown by the absence of Romberg's sign in the latter. Here, as the defect is not due to loss of those sensory components that control equilibrium, it cannot be in any way compensated by vision. The subject of cerebellar disease therefore does not become more unsteady when his eyes are closed.

Disturbances of Gait.—The disturbance of gait in cerebellar disease is one of its most prominent symptoms. If the disease is bilateral the patient staggers and sways about like a drunken person, in fact probably no other common simile in medicine is so justified as the term "drunken" gait (*démarche ébrieuse*) of cerebellar disease; the patient staggers and reels from side to side, deviates from his normal line of progression and recovers himself awkwardly; he raises his feet irregularly and fails to bring them to the ground in a normal relation to one another. At the same time the feet are kept widely separated so as to broaden the base of support, and the toes are generally directed outward by outward rotation of the limbs.

Unilateral cerebellar lesions produce an even more characteristic though less severe disturbance of gait. There is less difficulty in maintaining equilibrium, but the patient staggers, recovers his balance awkwardly, and shows a tendency to reel toward the side of the lesion.

FIG. 63



FIG. 64



FIG. 65



Further, there is an almost constant tendency, if the disease is acute, to deviate from the direct line of progression toward the side of the lesion, and it can be frequently observed that the patient attempts to compensate this by rotating his trunk on its long axis toward the opposite side, so that the shoulder of the affected side stands in advance of its fellow, as if he desired to deviate from his actual line of progression toward the normal side. The patient is often aware of this tendency to deviate toward the side of the disease; a patient with a tumor of the left lateral lobe of the cerebellum once told me: "I know I am always going to the left, but I can't help it. I feel as if something were pulling me over." Further, the affected leg is generally abducted and rotated outwards, and its movements are ataxic.

Speech.—Speech often presents characteristic alterations, generally, however,

only with extensive disease; it becomes slow, explosive, and scanning, and is often slurred. This seems to be a result of the incoördination of the mechanisms of phonation and articulation.

Ocular Movements.—But even more characteristic is an affection of the ocular movements. Ocular palsies are never a direct result of cerebellar disease, though they not infrequently occur in cases of tumor or abscesses owing to pressure transmitted directly or indirectly to the nuclei of the ocular nerves. Thus the sixth nerve suffers the most frequently, but tumors in the anterior part of the vermis may produce a palsy of the trochlear or of some part of the oculomotor nerve. Weakness of lateral conjugate movement of the eyes toward the side of the lesion is probably also only an indirect effect. The "skew deviation," originally observed by Magendie, seems, however, to be attributable to the disease of the cerebellum itself.

Nystagmus.—Nystagmus is a common and very valuable localizing sign of local cerebellar lesions. It is almost certainly a true cerebellar symptom, though Bing has regarded it as an effect of the pressure of the cerebellar disease on the abducent nucleus, or on the dorsal longitudinal bundle. It is rarely observed on direct forward fixation of the eyes, except with severe and acute lesions, and generally appears only on lateral or more rarely on vertical movements. When the cerebellar disease is unilateral the nystagmus is most marked on conjugate deviation of the eyes toward the side of the lesion, and consists of a relatively slow forcible jerking movement in this direction and a gradual recession toward the middle line. On conjugate deviation toward the opposite side the nystagmoid movements are more rapid, but smaller in range. The nystagmus is almost invariably conjugate, *i. e.*, the eyes move simultaneously and equally. Nystagmus is less commonly associated with vertical movements of the eyes, and when it occurs is generally rotatory, in the direction of the movement and toward the side of the lesion. Nystagmus is such a common symptom of labyrinthine disease that its differentiation must be considered. This is not easy, except by exclusion of vestibular disease, but Barany's observations tend to show that the suppression of calorific nystagmus associated with spontaneous nystagmus, indicates a central lesion in the cerebellum or pons Varolii.

Nystagmus is much more common in acute than in chronic cerebellar disease; it is rarely prominent in the slowly progressive atrophies.

Hypotonia.—Hypotonia is another true symptom of cerebellar disease, though it is usually prominent only in cases of acute lesions. Like the ataxia it is more or less limited to the limbs homolateral to the lesion. It shows itself in the unnaturally large range of passive movement; by the absence or diminution of the resistance that well-toned muscles offer to stretching, and by their flaccid consistence. If a hypotonic arm is held below the elbow and passively shaken the hand and fingers can be seen to flap about like the limb of a flail, quite unlike a normal limb.

Another sign of the hypotonicity was described originally by Holmes

and Stewart; the patient is asked to place his elbow on a table or other rest and to flex his forearm as strongly as possible, the observer resisting the movement by grasping his wrist. When the wrist is suddenly released the forearm rapidly flexes for a short distance, but if the limb is normal it is quickly brought to rest by the sudden reflex tonic contraction of the antagonists of the contracting muscles, *i. e.*, the triceps; and if the limb is spastic there may be a considerable recoil. In cerebellar hypotonia, on the other hand, the movement is not checked by this reflexly excited contraction of the triceps, and the forearm continues to move until flexion is no longer physically possible.

Paresis and Astasia.—Paresis or asthenia of the homolateral muscles has been emphasized by Luciani as an important sign of cerebellar disease, but it occurs in man only with acute and extensive lesions, and is usually only transient. The defective coördination and synergy of the affected limbs may easily give an erroneous impression of their strength. The affected limbs are often tremulous, and the head when unsupported may sway from side to side in small oscillations. This may be in part due to the deficient coöperation of those muscles which by their coördinated activity should maintain the steady attitude, but *astasia* or tremulousness seems to be a specific cerebellar symptom; it is due, as Luciani has shown by his experiments, to the failure of complete fusion of the individual muscular contractions. Patrizi has demonstrated it graphically.

Catalepsie Cérébelleuse.—Despite this tremulousness, which is so common in acute cerebellar diseases, certain patients show a remarkable ability to maintain attitudes as well or even longer than normal persons. Babinski has described this as *catalepsie cérébelleuse*. He tests it by placing the patient on his back, with his thighs flexed on his pelvis, the legs flexed at the knees and the feet separated; at first there is the usual pathological tremulousness, but the limbs soon become steady and may then remain immobile in this difficult attitude for a remarkable time. This is rarely a prominent symptom, and it seems probable that even the cases in which Babinski originally described it were not instances of pure cerebellar disease. Kleist has shown that this symptom also occurs in motor and ideomotor apraxia, and it may be consequently due to a lesion of the frontopontine fibers.

Adiadokokinesia.—A more valuable sign also described by Babinski is that which has become known by the term *adiadokokinesia*; it consists of the inability to execute rapidly repeated movement at a normal rate. It is best tested by making the patient flex his elbows to a right angle, and then pronate and supinate his forearms as rapidly as he can. If the cerebellar affection is unilateral the symptom is very prominent; the movements of the affected limb are slower, less regular, and less complete in range than those of the normal side, and as the attempt is persisted in they become wilder, so that eventually the patient's aim cannot be recognized in his efforts. In many normal persons this rapid succession of movements is not executed so well with the left arm as with the right, but when it is present *adiadokokinesia* is so prominent

that there can be no danger of mistaking it. This symptom, however, is not always present in even extensive disease of the cerebellum, nor is it pathognomonic of cerebellar lesions.

Vertigo.—Vertigo is usually described as a common symptom of cerebellar disease, but it is usually in cases of tumor or abscess of this organ that it occurs, and as the pressure these conditions produce is liable to affect the vestibular nerves or their pontine nuclei it is probable that the vertigo is not a direct effect of the cerebellar lesion. Still it must be admitted that it is often met with in cases of atrophy of the cerebellum, and may thus be regarded as a cerebellar symptom. Unhappily vertigo or the commoner English words "giddiness" and "dizziness," are applied to widely different conditions, so that it is always necessary to ascertain exactly what the patient denotes by them.

These terms are commonly applied indifferently to two states: either to an indefinite, usually indescribable sensation of insecurity or unsteadiness, with a feeling of faintness and confusion, or even a momentary obscuration of consciousness, and dimness of vision; or to a subjective movement of self or of surrounding objects, generally as a rotation about the longitudinal axis of the patient himself, associated or not with some actual movement. The former variety is of no localizing value; it may result from various causes and is common in all conditions of increase of intracranial pressure. True vertigo, on the other hand, is an important symptom in cerebellar and vestibular disease, but as it is entirely subjective it must be investigated with care. Some years ago in conjunction with Dr. Grainger Stewart I published records to show that in cases of unilateral cerebellar tumor the apparent movement of objects in front of the patient is from the side of the lesion toward the healthy side, and that the subjective rotation of self is in the same direction. With extracerebellar tumors, or tumors of the pontocerebellar angle, which always compress or involve the vestibular nerve, we found that the apparent rotation of external objects was in the same direction, *i. e.*, from the affected toward the healthy side, but the subjective movement of self in the opposite direction. Others have failed to confirm this relation, though I have been able to substantiate it in numerous cases. Occasionally the vertigo consists in an apparent vertical movement of self or of external objects; its localizing value is uncertain, but it is said to occur most frequently with tumors in the anterior vermis, possibly with such as compress the corpora quadrigemina.

Other Symptoms.—No form of sensation is affected by disease limited to the cerebellum; repeated observations have failed to show any defects of deep sensibility, as Lewandowsky's views would necessitate, or any diminution in the appreciation of weights, as Lotmar has described.

The superficial and deep reflexes are also unaltered in pure cerebellar disease, or the knee-jerks may be found slightly exaggerated. The sphincter functions too are unaffected.

Finally, the cerebellum is in no way concerned with the psychical

processes, and no component of mind is affected by its disease. It is true that idiocy or some form of mental deficiency is often observed associated with congenital or early acquired cerebellar disease, but this can be attributed only to disease of other parts of the brain.

Recovery of Cerebellar Symptoms.—It is a fact emphasized by all physiologists and confirmed by clinical observations that the early and severe symptoms which result from acute cerebellar lesions gradually diminish and may even disappear completely. The explanation of this common observation probably lies in the fact that these early symptoms are partly due to the shock of the acute lesion on other parts of the central nervous system closely related to the cerebellum, or to that dynamic factor which von Monakow has defined as diaschisis, a functional depression due to passive inhibition of centres intimately associated in function with that destroyed. As this diaschitic effect passes off the symptoms diminish in intensity. A further factor in the early improvement is the functional readjustment of the nervous mechanisms disturbed by the lesion: it is a well-known instance that though section of one vestibular nerve produces violent forced movements and disturbances of equilibrium, the immediate section of the opposite never diminishes these symptoms; it establishes a relative functional equilibrium. Similarly the gradual readjustment of the functional balance that a local lesion of the cerebellum has disturbed may materially diminish its symptoms.

But the amount of recovery is so great, and the amelioration of symptoms frequently so extraordinary that a further explanation must be sought. That which has been generally offered, and practically the only one considered by physiologists, is that the functions of the portions diseased are replaced or compensated by the vicarious activity of other centres, either in the cerebellum itself or elsewhere, and particularly by the forebrain. There can be no question but that one portion of the cerebellum can to some extent compensate the destruction of another portion; the fact that there is generally a certain relation between the intensity and permanence of the symptoms and the extent of the disease is evidence of it. There is also much in support of the hypothesis that the gradual amelioration of cerebellar symptoms is due to the compensatory activity of other organs; it has been repeatedly demonstrated, for instance, that symptoms of a unilateral cerebellar lesion recur or become more intense if the opposite cerebral hemisphere is injured, and Bickel and Jacob have shown that if in a dog which has partially reacquired equilibrium after a partial destruction of the cerebellum the posterior lumbar roots are cut the animal becomes more or less permanently unable to stand. Human pathology, too, illustrates the same fact, as it is recognized that recovery from the symptoms of a cerebellar tumor that is removable is less favorable if the case is complicated by hydrocephalus which has damaged the forebrain. We may thus conclude that the diminution of the symptoms of an acute destructive lesion of the cerebellum can be ascribed to at least three factors: the passing off of the effect of shock

or diaschisis which every acute lesion produces on functionally associated parts; to the readjustment of the normal functional equilibrium which was upset by the lesion; and to the vicarious compensation of the part destroyed by other parts of the nervous system.

Symptoms of Chronic Cerebellar Disease.—As those symptoms which we have seen supervene on acute cerebellar lesions gradually diminish with time, and may in part disappear, we must now consider the symptoms which characterize chronic or slowly progressive disease of the cerebellum. These are represented by the systemic atrophies and degenerations of the cerebellum, as well as by old softenings and hemorrhages, and by neoplasms that have become latent.

In these cases it frequently happens that the symptoms are much less marked than might be expected from the severity and extent of the disease.

The disturbances of movement are invariably the most pronounced, and it is especially gait that is affected. Though the individual and simple movements of the upper extremities may be executed fairly well, or may show at the most only a moderate degree of ataxia, the more complex acts which require the coördinated and synergic co-operation of many groups of muscles are generally grossly ataxic. Gait is consequently as a rule very severely affected, and may show all the characters of that described as a result of acute cerebellar lesions. The patient reels and staggers from side to side as a drunken man, and equilibration may be so much disturbed that support becomes necessary.

Yet the movements of the lower limbs as the patient lies on his back, or when fully supported, are not in equal degree ataxic; the difficulty in maintaining equilibrium and directing locomotion is consequently due not so much to the ataxia of the legs as to a disturbance of the synergia of the muscles of the legs and trunk which is necessary for balance.

But the limbs never escape completely; the finer movements are at least awkward and slow, and often show signs of asynergia and dysmetria; Babinski's *adiadokokinesia* can be usually demonstrated. In certain forms of cerebellar atrophy, tremor, when the limbs are unsupported or even during motion, is a prominent symptom, and oscillation of the head is common in the severer cases. When the patient stands erect, or even as he sits more or less unsupported, the body may be seen to sway about irregularly. Occasionally even the movements of the face, both volitional and expressional, are ataxic and dysmetric; while speech is even more frequently affected than in acute cerebellar disease, being slow and drawling, or explosive and scanning. Nystagmus is, on the other hand, not so common or so characteristic. Vertigo is a frequent symptom; it is usually of the indefinite variety, and not accompanied by any definite sense of rotation of self or of external objects. In the sudden attacks the patient may lose equilibrium, and if without support may fall.

Hypotonia, too, has been frequently described as a symptom of chronic

cerebellar disease, but though it is undoubtedly, as a rule, present, it is in my experience rarely marked or prominent. The affected limbs are usually feeble in relation to their muscular development, but local palsies or pareses do not occur.

The reflexes are unaltered, unless their normal state be disturbed by associated lesions of other parts; and sensation remains unaffected.

Functional Localization.—One of the most important, and one of the most difficult questions on which we must touch is the value of the symptoms hitherto described in the localization of disease in the cerebellum; on the answer to this depends the success with which surgical intervention is possible in those forms of disease which are amenable to surgical treatment.

If the symptoms of lesions in different portions of the cerebellum differ it is necessary to assume the separate localization of certain functions in the cerebellum. We must consequently refer shortly to any evidence there is of functional localization in this organ. This question can be considered from different points of view—in the first place we might ask if any of the essential symptoms of cerebellar disease, as ataxia or atonia, results from a lesion of one region of the cerebellum only; or secondly, if any portion of the cerebellum is concerned more or less exclusively with the control of any group of muscles, or even of any part of the body, as of an arm or of the eyes. In this discussion it would be desirable to consider the cortex and the central nuclei separately, but we know as yet so little of the functions of the latter that they cannot be profitably discussed. For years Luciani's view that the cerebellum is a functionally homogeneous organ and that all parts of the cortex are functionally equal, has held sway. It is true that many physiologists, and especially Ferrier, obtained different motor effects from the electrical stimulation of different regions of the cortex; but it was generally believed, and has been now shown, that these motor effects were largely, if not entirely, due to spread of the current to neighboring parts: Horsley and Clarke have proved that the cerebellar cortex is relatively inexcitable. Recent work, especially by the Dutch anatomist Bolk, has again raised interest in the question. From an extensive study of the comparative anatomy of the cerebellum he came to the conclusion that certain regions of this organ are developed in relation to the activities of certain parts of the body, and therefore assumed a functional correlation between them. Shortly stated, he believed that the unpaired part of the cerebellum—the vermis—controls bilaterally synergic movements, as those of the trunk, and of the limbs in gait, while each lateral lobe is the centre for the control of simple and independent homolateral movements.

This view has been further elaborated by van Rynberk and others; according to one of their schemes the muscles of the head, eyes, and larynx are represented in the anterior vermis, next in order posteriorly the muscles of the neck and the bilaterally innervated movements of the limbs, and in the posterior vermis the muscles of the trunk.

By the anterior portion of the lateral lobes the unilateral movements of the upper extremities are controlled, and the lower limbs by their posterior halves. These views have received some support from the experiments of van Rynberk, Luna, Pol, Rothmann, and others, but their results have not been sufficiently uniform to admit a general conclusion. Clinical observations in man have not yet tended to confirm this theory of strict localization of function in the cerebellum, and interesting though it may be, and possibly important for future work, we must conclude that as yet it has no clinical significance.

On the other hand, clinicians from the time of Nothnagel's observations (1876) have been inclined to ascribe considerable differences to the symptoms evoked by disease of the lateral lobes and of the vermis, and some have even asserted that recognizable symptoms are due to affection of the latter only. But though this view can certainly not be maintained there is no doubt that a lesion of the vermis generally produces more disturbance than one of equal size in the lateral lobes, and it usually shows itself more prominently in the affection of equilibrium. There seems much justification for Thomas' assertion that the vermis is especially concerned in the maintenance of those coördinations on which the equilibrium of the body depends, while the lateral lobes regulate the individual and more specialized movements of the limbs.

This view conforms with the chief anatomical connections of each part; it is the vermis that receives the afferents from the vestibulum, the organ of equilibration, and the direct spinocerebellar tracts, while the chief connection of the lateral lobes is with the forebrain which controls and regulates the more specialized movements of the limbs.

Finally, the most important clinical application of the doctrine of cerebellar localization is that unilateral lesions of the cerebellum produce predominantly homolateral symptoms, and the more strictly unilateral a lesion is, and the less it invades the vermis, the more strictly will the symptoms be homolateral.

Disease of the Cerebellar Systems and Connections.—Following our scheme we should now consider what are the special symptoms that result from lesions of the chief connections of the cerebellum, but unhappily our knowledge is here so incomplete that only a short summary can be given. It has been already pointed out that Marburg and Bing obtained motor disturbances more or less similar to those produced by cerebellar lesions by section of the spinocerebellar tracts in animals; and their degeneration in man, as in Friedreich's disease, produces very similar symptoms. Unhappily degeneration of them alone has been very rarely observed; in Friedreich's disease it is associated with degeneration of the dorsal columns and of the pyramidal tracts. However, observations tend to show that, as distinguished from the symptoms of lesions of the cerebellum itself, the degeneration of these tracts produces less disturbance of equilibrium and less muscular hypotonicity in relation to the amount of incoördination: but this ataxia is very similar to that of cerebellar disease,

in that it is seen chiefly in the larger and more complex movements. No other spinal tract connected with the cerebellum is liable to be affected alone.

It is only in the corpus restiforme that the various cerebellar connections are liable to suffer, uncomplicated by destruction of other tracts, in their passage through the bulb. They are generally involved here by softenings—as that which results from lesions of the posterior inferior cerebellar artery, or by tumors. The immediate symptoms are generally severe and gradually diminish with time. The most pronounced are ataxia of the homolateral limbs, a gait reeling and staggering chiefly to the side of the lesion, and marked disturbance of equilibrium due probably to involvement of the vestibular nerve or its nuclei or of Deiters' nucleus. But it is only by the associated symptoms due to disturbance of other centres, or tracts, that lesions of the corpus restiforme can be safely diagnosed.

Isolated disease of the cerebropontine tracts is unknown, though Thomas has found them degenerated in one of his cases of olivo-ponto-cerebellar atrophy. The middle cerebellar peduncles, however, are occasionally affected alone, as by a softening in their course or in the ventral half of the pons. The symptoms such lesions produce seem to differ from those due to a homolateral lesion of the cerebellum chiefly in that there is little disturbance of equilibrium though considerable ataxia, and the unsteadiness and tremulousness of unsupported parts of the body is usually very pronounced. In one case I have seen the irregularity of voluntary movements belonged rather to the intention-tremor type.

Forms of Cerebellar Disease.—We must consider what are the chief forms of cerebellar disease. From the point of view of treatment cerebellar disease may be divided into two main classes: (1) Intrinsic or essential diseases of the cerebellum, as the cerebellar atrophies and degenerations, and defects due to congenital lesions, or to a hypoplasia of the whole or part of the organ. (2) Cases in which the cerebellar disease is due to some extrinsic lesion, for example, its involvement by a tumor, or abscess, or by hemorrhage or a necrotic softening due to vascular disease. In this class, too, we must consider the disturbances of function due to compression of the cerebellum by a rise of intracranial pressure, such as occurs in hydrocephalus.

The first class, that which includes the intrinsic or essential diseases of the cerebellum, is a large and composite group, and the classification of the types included in it is difficult; such an attempt at classification as the following will, however, make our subsequent descriptions more easy.

1. Agenesis or congenital hypoplasia of the whole or of part of the cerebellum. Many of these cases show no signs of functional derangement, but they are sometimes associated with idiocy or epilepsy.

2. Primary parenchymatous degeneration of the cerebellum, either general or local, but limited to the cerebellum (Fraser, Holmes). It is usually hereditary or familial.

3. Primary hereditary or sporadic cerebellar degenerations associated with disease of other parts of the nervous system, either of the spinal cord (as occasionally in Friedreich's disease); of the brain-stem (Thomas' olivo-ponto-cerebellar type); or of the forebrain.

4. Cerebellar degenerations secondary to toxic influences, or meningeal or interstitial changes.

In the first three subgroups the disease is due to a congenital deficiency, or a congenital or hereditary tendency to degeneration of all or of certain of the functional elements of the cerebellum, and treatment can neither remove the disease when it is developed, nor check its development. The sole aim of therapeutics, therefore, can be to ameliorate the symptoms which result from this disease.

In the fourth subclass, that including cerebellar degenerations due to toxic, interstitial, or meningeal changes, therapeutics may arrest the development of the disease or even remove it. But these cases unhappily form only a small proportion of the cerebellar degenerations. Rossi and Murri have observed cases in which a primary degeneration of the cerebellar cortex set in after attacks of enteritis, and was due apparently to toxins of intestinal origin. In such cases the first aim of treatment would evidently be the removal of the cause, or if its nature could be determined an attempt to neutralize it.

Primary interstitial disease and secondary involvement of the cerebellar elements may be illustrated by Schultze's case, in which a progressive sclerosis of the cerebellum was attributed to chronic alcoholism, or by the cases of Michell Clarke and Catola, in which diffuse foci of sclerosis were found throughout the cerebellum, as well as elsewhere in the central nervous system. In certain of the senile atrophies it seems probable that the disease is secondary to arteriosclerosis. Occasionally this type of disease is of syphilitic origin; in Southard's case, for instance, the atrophy of the cortex was associated with, probably in the relation of effect and cause, chronic congenital syphilitic leptomeningitis. In one of Vogt's and Askwatzturow's cases, too, the disease was probably secondary to a meningitis. In such cases there is no doubt that if the primary disease be syphilitic early and adequate treatment on the lines laid down in the chapter on the Treatment of Syphilitic Disease of the Nervous System would be effective. The great probability is that in most of these cases the meningitis is syphilitic, and as it is the form of disease which is most under our control all possible efforts should be made to confirm any suspicions there may be by the Wassermann test applied to the blood and the cerebrospinal fluid, and by the chemical and cytological examination of the fluid.

Other forms of meningitis, however, may produce structural changes in the cerebellum or the symptoms of cerebellar disease. I have seen diffuse atrophy of the cerebellar cortex subsequent to a posterior basic meningitis. On the other hand, as Lüthje has pointed out, the majority of those cases of acute ataxia which come on during or after infective diseases are not of purely cerebellar origin, but are more frequently due

to diffuse cerebrospinal lesions; in Ebstein's case, for instance, it could be attributed only to diffuse meningeal and perivascular lesions throughout the spinal cord and medulla oblongata. Finally it is now known that acute cerebellar ataxia may result from involvement of the cerebellum by acute poliomyelitis or polioencephalitic lesions; and unhappily when these develop treatment can be directed only to a possible amelioration of the symptoms.

We must now consider what treatment can effect in those cases in which we are unable to remove the disease or influence its course. The reply is disappointing, for it must be admitted that of all the symptoms of nervous disease probably none is so little influenced by therapeutics, medicinal or mechanical, reëducative or surgical, as the motor disturbances of cerebellar origin, and these are usually the predominant symptoms with which the patient comes under observation. Drugs are useless, and the symptoms do not depend on paresis of the muscles, or contractions or rigidity such as might be influenced by massage, active or passive movements, or hydrotherapeutics.

The brilliant results obtained by the employment of Frenkel's reëducative exercises in the treatment of tabetic ataxia have probably repeatedly raised vain hopes that by them the disturbance of gait and the incoördination of movement in cerebellar disease might be relieved, but an analysis of the nature of the defect in these two types of disease must show that such hopes are vain. For while tabetic ataxia is dependent chiefly on loss of the peripheral, sensory and non-sensory, afferents which control movement, and can be consequently relieved by teaching the patient to replace these by vision, the ataxia, or a synergia and dysmetria of cerebellar disease, results from a disturbance of the central coördinating mechanism, on the functional integrity of which the accuracy and precision of movement depends. And experience has confirmed this *a priori* argument; in my own experience, and it agrees with that of others, little manifest improvement follows the most careful and persistent attempts to train chronic and stationary cases with recent cerebellar ataxia, and the diminution that may gradually occur in the symptoms of recent cerebellar lesions is only that which may be expected independent of the training. But proper supervision and care is not wholly useless in such cases. It is in the first place usually evident that the intensity of the symptoms depends largely on the general health and strength of the patient; when he is run down or in any way exhausted he has more trouble in carrying out the movements than when in good health. It is consequently always advisable to maintain his general health at as high a level as possible.

Further, when the patient is tired or exhausted his symptoms become more pronounced, and there is evidence that the stress of exertion or overwork may lead to even a permanent increase in the symptoms.

Vertigo, alone or associated with tinnitus, is often one of the most distressing symptoms; it seems to be most frequent in the senile atrophies which are associated with arteriosclerosis, and can be often

relieved by the treatment of this condition. I have found moderate doses of iodides and bromides combined with arsenic effective.

But if therapeutics can do little for the intrinsic atrophies and degenerations of the cerebellum it is happily often otherwise with that group of cases which I have included under the term extrinsic diseases—that is, cases in which the symptoms are due to the involvement or compression of the cerebellum by a tumor or an abscess, or by a general rise of intracranial pressure. In such cases surgical intervention frequently yields brilliant results, but these depend in the first place on the early diagnosis and the accurate localization of the disease. As the surgical treatment of these cases is dealt with in another chapter we are concerned here only with the principles on which diagnosis and localization of the disease depend.

Cerebellar Tumor.—Gliomas are the most common tumors of the cerebellum at all ages. These are usually very cellular and grow rapidly, though not infrequently they have an extraordinarily slow course. Many unhappily infiltrate the tissue around them widely, and if they start in the lateral lobe are liable to extend into the middle cerebellar peduncle, or even into the pons or medulla oblongata. Frequently they undergo central necrosis with the formation of a cyst, which by the accumulation of degeneration products and fluids within it may compress the actively growing tumor so severely as to inhibit or check its further growth. Occasionally when such a cyst is examined post mortem it is found with sclerosed walls and with but little malignant growth around it. As gliomata tend to infiltrate rather than destroy the tissues they may produce, even when large, general symptoms of intracranial pressure without pronounced local symptoms. And as they infiltrate widely their surgical treatment is usually unsatisfactory, as complete removal is often not possible and consequently recurrence is likely.

Tuberculous tumors are probably the next most common, especially in childhood. They usually produce distinct symptoms, but the danger of disseminating the tubercular disease through the meninges makes them unfavorable for surgical measures. Often, however, they seem to become latent and then their symptoms gradually diminish. Gummata in the cerebellum are relatively uncommon considering the frequency of cerebrospinal syphilis, and I have only rarely seen malignant metastases involve the cerebellum.

Though very rare in Great Britain echinococcus and cysticercus cysts occur frequently on the continent of Europe or elsewhere. Simple cysts also occur and are probably the most favorable of all tumors from the surgical point of view; their pathology is obscure, but it seems probable that they are either congenital cavities which only produce symptoms in later life by the accumulation of fluid within them; or they may be due in the first place to the adhesion of folia of the cerebellum together by a slight meningitis and the formation of a retention cyst in the sulcus between them.

Not infrequently the symptoms of a tumor of the cerebellum are due

to its compression by a growth outside it. Endotheliomata of the dura mater of the posterior fossa or osteomata may compress the cerebellum, but the most common cause is a fibroma or neurofibroma of the cerebellopontine angle, growing from one of the cranial nerves. The majority of these tumors arise in connection with the eighth nerve, and are merely fibromata of the same structure as the so-called neurofibromata of other nerves. They frequently undergo myxomatous or other degenerative changes in places. Less commonly they are found attached to the fifth or the seventh nerve. They are occasionally part of a general neurofibromatosis, and may be bilateral, as in the cases described by Henneberg and Koch and Stewart and Holmes.

A rarer form of tumor which may compress the cerebellum is a cholesteatoma. This usually develops from the middle ear and extends inward along the internal auditory meatus to the posterior fossa.

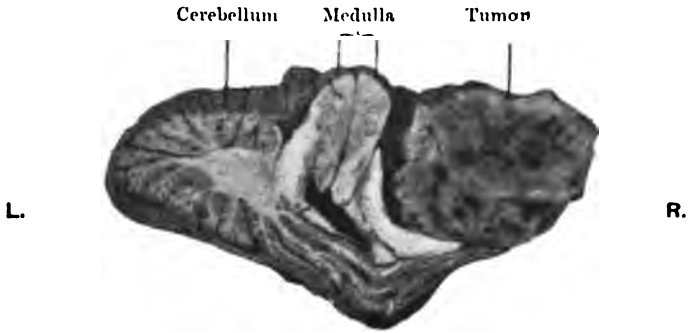
Symptoms.—As a rule careful examination will reveal symptoms as definite and characteristic as those produced by tumors in any other region of the nervous system. These may be divided into the general symptoms of intracranial tumor, the local or direct symptoms, and the accessory symptoms due to direct or indirect affection of neighboring parts. (Plate XLVIII, Fig. 1.)

GENERAL SYMPTOMS.—The general symptoms are those due to the pathological increase of intracranial pressure. Headache is almost inevitable, and is often extremely severe and persistent. It is generally most intense in the occipital region and often is described as spreading down the back of the neck; it may be associated with a local tenderness of the skull to pressure or percussion. Occasionally the headache is chiefly frontal or vertical. Vomiting, too, is almost invariably present; but it differs in no way from that which occurs with other intracranial growths. Optic neuritis is almost constant; personally I have not seen a case of intracerebellar tumor in which it was not present when the patient first came under observation, and its absence has been very rarely noted. The amount of swelling and exudation is probably on the average greater than with supratentorial tumors, and loss of vision may set in early. Its greater intensity in one eye cannot be accepted as a reliable guide to the side of the cerebellum involved by the tumor.

LOCAL AND DIRECT SYMPTOMS.—The local or direct symptoms of cerebellar growth are those of local cerebellar disease which we have already dealt with; here we can only refer to them as signs of value in the exact localization of the site of the tumor. As a rule, the more rapid the evolution of the tumor, and the more destructive it is, the more pronounced and definite are the symptoms. But it is also in such cases that the physical signs are often obscured or complicated by the indirect pressure which the tumor exerts on portions of the cerebellum it does not involve, or on neighboring organs in the brain-stem; for instance a tumor that involves only one lateral lobe of the cerebellum may disturb the functions of the vermis, or even those of the opposite lateral lobe, as well as those of the pons and medulla.

PLATE XLVIII

Fig. 1



A Tumor of the Cerebellopontine Angle (Fibroma of the Eighth Nerve) Compressing the Cerebellum and Brain Stem. (Holmes and Stewart.)

Fig. 2



"Skew Deviation" after Removal of a Tumor from the Left Lateral Lobe of the Cerebellum: the left eye is directed downward and inward, the right eye upward and outward. (Holmes and Stewart.)

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Ataxia.—Ataxia is usually the most prominent symptom; when the tumor lies in one lateral lobe this is exclusively or predominantly present in the homolateral limbs. If the vermis is involved it is bilateral and as a rule relatively greater. It is more marked when the lesion involves one of the peduncles, especially the inferior, than when it is superficial. If the superior peduncle be affected the disturbance of movement frequently approximates to the intention-tremor type rather than to the characteristic ataxia. Apart from this it cannot be said that the ataxia is a guide to the exact site of the lesion. As in all acute lesions the disturbance of equilibrium in gait is more prominent than the ataxia of the simpler movements; in unilateral tumors the patient reels and staggers to the side of the lesion, but this rule is not absolute as the reeling occasionally is chiefly toward the sound side; this seems to be due mainly to a volitional attempt of the patient to compensate the defects the lesion produces by shifting his centre of gravity as far as possible toward the healthy side. In tumors of the vermis or those of bilateral extension gait is usually much more impaired. From my own observation it seems probable that tumors in the anterior portion of the cerebellum produce less disturbance of coördination and equilibrium than those situated posteriorly.

Frequently unilateral tumors produce a more or less characteristic attitude of the head; it is generally rotated with the chin toward the healthy side and at the same time flexed toward the affected side, so that the occiput approaches the shoulder on the side of the tumor.

Hypotonia and paresis seem to be more pronounced when the inferior peduncle and the central nuclei are involved.

Nystagmus.—Nystagmus is one of the most valuable symptoms; as described above it is usually apparent only on lateral movement of the eyes, and the conjugate excursions are slower, more forcible and wider in range, on movement toward the affected than to the healthy side. Nystagmus on vertical movements of the eyes suggests a lesion in the anterior portion of the cerebellum and is thus a valuable localizing symptom.

Vertigo.—Vertigo is certainly much more common and of a more definite type in lesions of the postero-inferior portions of the cerebellum than elsewhere; this is probably due to the fact that it results chiefly from disturbance of the vestibular connections. True vertigo with a distinct sense of rotation of self and of surrounding objects is in my experience rare with tumors in other parts of the cerebellum. Despite the lack of confirmation by other clinicians I am convinced of the importance of the directions of the apparent rotations as a differential sign in the diagnosis of unilateral intracerebellar and pontocerebellar tumors; in the former the movement of both self and the external world seems to be from the affected toward the healthy side, but in the latter class, self seems to rotate toward the affected side, but external objects from the affected to the normal side.

ACCESSORY SYMPTOMS.—But it is largely on the accessory symptoms that we must rely in the localization of the site of the lesion. In this

respect ocular palsies are important. Disease limited to the cerebellum cannot produce directly paralysis of any ocular muscle, or palsy of any movement; in cases of cerebellar tumor, however, palsies are not infrequent, for they result either from a general rise of intracranial pressure, or from compression of those parts of the brain-stem that contain the ocular nuclei. Isolated palsy of one external rectus is most commonly due to a general rise of intracranial pressure, and, as is well known, frequently occurs with this condition no matter what its cause is. It is consequently of no localizing value. An external rectus palsy may be, however, due to compression of the sixth nerve by a tumor of the cerebellopontine angle. A conjugate weakness of lateral movement of the eyes, on the other hand, results from compression of the homolateral sixth nerve nucleus by a tumor in the postero-inferior portion of the cerebellum, or possibly from interference with a supra-nuclear centre for conjugate movement in or near Deiters' nucleus. Vertical movements are very rarely affected by intracerebellar tumors, but occasionally a weakness of upward movement results from the compression of the midbrain by a tumor that lies far forward in the cerebellum. I have never seen the pupillary reaction disturbed.

The so-called "skew deviation" of the eyes, the eye on the side of the lesion being directed downward and inward, the opposite outward and slightly upward, seems to occur chiefly with acute lesions that involve the cerebellum in the region of entrance of the middle and inferior peduncles. (Plate XLVIII, Fig. 2.)

A slight inferior neuronc weakness of one side of the face is occasionally observed when a large tumor lies low in the cerebellum and compresses the pons; a greater weakness is usually due to extension of the tumor into the pons, or to direct compression of the facial nerve by a tumor of the cerebellopontine angle. When a tumor lies far laterally in the cerebellum the trigeminal nerve may be compressed, but, as a rule, only its sensory portion suffers; this usually produces only pain or paresthesia on the same side of the face, actual sensory loss being rare. The corneal reflex, however, is often lost very early. Intracerebellar tumors and tumors involving the pons, however, frequently produce complete or considerable anesthesia in the distribution of the trigeminal nerve, with weakness or paralysis of the muscles innervated by its motor root. This is consequently an important sign in the differential diagnosis of these different conditions.

Disturbances of hearing are very important; while more or less complete unilateral nerve deafness is the rule in extracerebellar tumors, any degree of deafness is rare with intracerebellar growths. Tinnitus, on the other hand, is common in cerebellar tumors, but it is rarely so persistent or severe as with tumors of the cerebellopontine angle.

Affection of the functions of the glossopharyngeal, vagus, and hypoglossal nerves is very exceptional in intracerebellar disease, but some weakness of the same side of the palate is not uncommon with tumors of the cerebellopontine angle.

The Differential Diagnosis of Cerebellar Tumors.—The actual recognition of a cerebellar tumor is usually easy if proper regard be paid to the signs and symptoms which it produces, though it occasionally happens that a tumor in this organ gives rise to general rather than local intracranial symptoms. On the other hand it must be admitted that conclusions as to the exact site of the tumor are often uncertain and unreliable in the present state of our knowledge.

The most important point in the differential diagnosis is the distinction of tumors involving the cerebellum from those which lie without it but compress it, from those that involve the brain stem, and from supratentorial growths.

The extracerebellar tumors, more particularly those which grow from the auditory or trigeminal nerves, from the structures in the lateral recess, or from the meninges in this region, are distinguished by the early and severe involvement of the cranial nerves, more particularly the fifth, eighth, and the glossopharyngeal; by symptoms of compression of the brain stem, especially contralateral hemiparesis and alteration in the reflexes; and by the slightness of the symptoms of cerebellar disturbance in relation to the other symptoms.

Tumors of the brain-stem, and especially of the pons, may simulate cerebellar tumors when they involve the cerebellar peduncles, but they generally produce some hemiparesis with the characteristic alterations of the reflexes, and sensory changes, especially a crossed hemianesthesia; in this condition only the appreciation of pain and temperature may be disturbed. Further, tumors of the brain-stem produce more extensive and more complete cranial nerve palsies, and especially dysphagia or dysarthria, while the general symptoms of intracranial tumor, and especially optic neuritis, may be less prominent or even absent.

Tumors of the fourth ventricle, growing either from its lining membrane or from the choroid plexus, have been frequently confused with cerebellar tumors; but they rarely produce pure and isolated cerebellar symptoms. Cranial nerve palsies are common, especially a nuclear sixth palsy; and bulbar symptoms may occur early. Vertigo has been the chief symptom in some cases, and sudden death in an attack of dyspnea is liable to occur. Bruns described as pathognomonic of free cysts in the cerebellum sudden attacks of intense headache, vomiting, and vertigo, which occur on sudden movement of the head.

The differentiation of cerebellar growths from those of supratentorial site is usually easy; in my experience the most common mistake has been the confusion of frontal tumors with cerebellar tumors in cases where there has been a great increase of intracranial pressure; but as a rule the absence of nystagmus, ataxia, and diadokokinesia is sufficient to prevent error.

Many cases of pseudo-tumor cerebri have presented symptoms of cerebellar disease and, as in a case of my own, this led to a wrong diagnosis—but no reliable differential signs are at present known. The

condition of meningitis serosa, especially associated with middle-ear disease, may produce similar symptoms, but they usually subside on the proper treatment of the local disease.

Internal hydrocephalus, too, may be confused with a cerebellar tumor; its longer course, however, the frequent predominance of early atrophic over neuritic changes in the optic disks, the evidence of general increase of intracranial pressure out of proportion to the local symptoms, and signs of involvement of the pyramidal tracts are usually sufficient to distinguish the two conditions.

The diagnosis of the nature of the tumor is always uncertain, or at the most only probable. Tuberculous growths are the most frequent in childhood and the presence of other tuberculous lesions may make their existence probable. A history of syphilis and a positive Wassermann reaction probably indicates a gumma. Otherwise the probability of a glioma is great, as this is the most common neoplasm in the cerebellum. An abscess is indicated by its rapid evolution; by the coexistence of foci of infection, especially middle ear or mastoid disease, and by the frequent evidence of an infective process at the onset, with rigors, fever, local headaches, and general malaise. The extra-cerebellar tumors, especially those that grow from the cranial nerves, are usually benign.

Treatment of Tumors and Abscess of the Cerebellum.—This is the question which concerns us most closely, and to which the previous pages have only led up. Yet the answer is on the whole simple, for as we cannot, except in rare cases of gummata, control or check the growth of the neoplasm we are forced to resort to surgical measures. But for successful surgical intervention accurate localization is the first essential, and it is perhaps more important in the cerebellum than elsewhere in the nervous system, as, owing to the proximity of the delicate vital centres in the bulb, it is necessary to limit casual exploration as far as possible.

It must not be understood, however, that surgical intervention is to be recommended immediately on the diagnosis of cerebellar tumors. With tuberculous lesions, for instance, the danger of the dissemination of the tuberculous infection is great; and, on the other hand, many of these tuberculomas cease to grow and become latent, their symptoms disappearing or diminishing as fully as could be expected after their removal and the further injury to the cerebellum the surgeon must produce in the operation. A further reason against operation if it can be avoided is the fact that tuberculous tumors are frequently multiple. I know some patients in whom years ago the almost certain diagnosis of cerebellar tumor was made, and who now present few or no symptoms of it. Too often, unhappily, the rapid progress of the case, and a threatened failure of vision from optic neuritis forces the physician's hand.

If a gumma is probable vigorous antisymphilitic treatment should be adopted in the first place. An injection of salvarsan may be tried, but should be followed by mercurial inunctions alternated with large

doses of iodides; the general experience at present is that salvarsan has but little influence on gummata when they are already developed. Apart from the greater safety of medicinal over the surgical treatment of gummata is must be remembered that these are frequently associated with a considerable chronic syphilitic meningitis which is not amenable to surgery.

Abscesses usually demand immediate surgical measures, but if their course is not very acute it is often advisable to wait, in the hope that they may become encapsuled and until the virulence of the infection diminish, as otherwise the danger of the development of a general septic meningitis is great.

Simple and hydatid cysts of the cerebellum are the most favorable for surgical treatment; a considerable proportion of the successful operations have been in this class of case. When the tumor is a glioma unhappily only operation can give relief, and yet the proportion of cases in which the relief is at all permanent must be very small, as complete extirpation is rarely feasible, and the danger of recurrence is consequently great.

The various forms of extracerebellar and lateral recess tumors can be dealt with only by the surgeon, and as they are usually benign the results may be exceedingly good; the danger of the operation to life is, however, great.

The technique of the surgical treatment of tumors and abscesses of the cerebellum is dealt with elsewhere and needs no reference here, but we may with advantage consider the prognosis in cases in which operation is necessary. Unhappily the desirable statistics are not available, as there is no doubt a general tendency to publish cases in which the removal of the tumor has been successful, without reference to failures.

In my paper with Grainger Stewart we published the records of 11 cases of intracerebellar tumor which were operated on, and 10 cases of extracerebellar tumor. In the former group 5 cases died soon after the operation, 3 at least died after the paper was published, from recurrence of a glioma in 1 case, with tuberculous meningitis in another, while in the third death was probably independent of the cerebellar disease. Two cases of simple cyst survive, and possibly 1 after the partial removal of a glioma. Of the 10 extracerebellar tumors 7 cases died immediately or soon after the operation, 1 several months later with a tumor on the opposite side, while 2 survive.

Of 25 of Oppenheim's cases which were operated on 3 recovered, 3 improved, while the others died. In 31 of the cases collected by Duret death occurred immediately or within some days after the operation; in 9 others death occurred later; 8 improved, while 5 recovered. Cushing's results have been much more favorable; of 35 operations 3 proved fatal, in 2 cases the tumor was inoperable, in 17 only decompressive operations were attempted, while he had 13 successful cases, tumor extirpations or cyst removals.

Pascalis has recently collected statistics on the operative treatment

of extracerebellar or pontocerebellar tumors. Of 11 cases in which only a decompression was attempted 3 died, and in 7 some improvement of the symptoms occurred; while in 101 cases in which removal was attempted 72 deaths occurred.

The promise of surgical intervention is thus not very favorable, but with the increase of accuracy in diagnosis, the more careful selection of cases, and the improvement which experience must evolve in surgical technique, the results of the future will be certainly better. Further, the fact must be taken into consideration that the majority of cases of cerebellar tumor must, if unrelieved, progress to a fatal termination and that consequently operation often offers the only chance of recovery.

But if an attempted removal of the tumor offers only such a poor chance of recovery it must be remembered that decompression alone often prolongs life and leads to a remarkable diminution of the symptoms. I know cases in which life has been prolonged for months, and even for more than a year in relative comfort. Experience shows that the most successful operation is bilateral occipital decompression by Cushing's method, in which a large portion of the skull is removed on both sides.

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CHAPTER XV

PARALYSIS AGITANS AND MULTIPLE SCLEROSIS AND THEIR TREATMENT

By CARL D. CAMP, M.D.

PARALYSIS AGITANS

PARALYSIS agitans (shaking palsy; Parkinson's disease; Schüttellähmung or Zitterlähmung) has been recognized as a clinical entity since first described by Parkinson, in 1817, although cases of similar description, and doubtless the same disease, were described as early as 1766, by Schwarz. It was not until a much later date that this affection was distinguished from certain types of chorea, multiple sclerosis, senile and hereditary tremors, and tremors due to lesions in the midbrain.

Paralysis agitans is not a very common affection; by the majority of observers the frequency is put at about 1 per cent. of all nervous diseases, though the observations of individuals vary from 0.2 per cent. to 1.5 per cent. The disease more often affects men than women and, judging from the statistics published, is more frequent in people living in country districts than in those from large cities. It usually begins between the fiftieth and sixtieth years of life, though cases beginning as early as the twentieth year or earlier are reported but are exceedingly rare.

Etiology.—A direct heredity occasionally occurs, more frequently there is a history of neuropathic disturbance or of rheumatic tendencies in the family, usually, however, the family history is entirely negative, and, as Mendel points out, the patient may show an exceptionally healthy and long-lived ancestry. It would seem that paralysis agitans affected mostly those persons whose lives had been devoted to hard work, either mental or physical, and attention has been called to the fact that there is a very small proportion of alcohol or tobacco users among cases of paralysis agitans (Dana, 10 per cent. smokers, 4 per cent. drinkers, among 127 male patients), nor can one trace any sexual or dietetic excesses to these patients. A history of syphilis is rarely obtained, and when it is present there is no benefit obtained by the use of antisyphilitic drugs. On the other hand, mental shocks and worry may be a potent cause and their persistence may hasten the progress of the disease. Physical overstrain also plays a distinct part in the production of the disease. A history of injuries to the part earliest or most involved is frequently obtained; these may have been severe or slight and of various kinds,

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such as burns, electric shocks, etc. The injury may have been one which the patient merely dreamed had occurred, as in a case mentioned by Mendel. A combination of injury and psychic shock is frequently an apparent cause. Continued exposure to cold and dampness has a bad effect on the disease and apparently causes it in some instances. In a few cases the onset of the disease has followed attacks of acute articular rheumatism or influenza.

Pathology.—Many efforts have been made to discover the lesion responsible for the symptoms of paralysis agitans. Repeated examinations of the nervous system have yielded conflicting results. The theories that explain the symptoms as the results of lesions in the cerebral cortex or in the midbrain have not been confirmed by histological examinations of these regions. Redlich and others have attributed the symptoms to islets of gliosis in the white matter of the spinal cord, while Dana and other observers have claimed that changes in the cells of the anterior horns of the grey matter of the spinal cord caused an interruption of the centrifugal motor impulses. There are many cases on record in which the examination of the central nervous system has shown it to be entirely normal, notably those of Schwenn, and Idelsohn. Camp, in 1907, examined, histologically, the central nervous system from 14 cases and could find no constant change, nor any which was peculiar to the disease. It seems most probable that the cause of the muscular rigidity is to be found in a change in the muscles themselves; such changes being described by Blocq, Schwenn, Shiefferdecker, Idelsohn, Camp, and others. The tremor is probably due to the rhythmic nerve impulses which, in normal persons, keep up the muscle tonus, but which, acting in a diseased muscle, produce a tremor. It is of prime therapeutic importance to know what initiates the change in the muscle, but on this point we can only theorize. The change may occur as the result of a congenital tendency such as apparently is the case in some of the muscular dystrophies, or it may be the result of some toxic influence on the muscle. So far no such toxic substance has been discovered. The secretions of the ductless glands have been carefully considered in this connection and there is suggestive evidence that an alteration or diminution of the secretion of the parathyroid glands may be the cause of the disease (Lundborg, Berkley, Camp, Alquier, and others).

Symptoms.—The most important symptoms, from the point of view of the diagnosis and, in most cases, from the therapeutic standpoint, are the stiffness or rigidity of the muscles, and the tremor. Of these two the former is perhaps the more essential, since a number of cases have been reported without tremor; the so-called *paralysis agitans sine agitatione*. Many of the other symptoms described as characteristic of the disease are a consequence of these two symptoms.

Muscular Rigidity.—The rigidity is usually described as lead-like. It may be general or affect only a part such as an extremity; usually the face is involved. The muscles affected do not atrophy but have a peculiar feel, as if one pressed on dead instead of living tissue. The

PLATE XLIX



Position of Paralysis Agitans.

Note position of the arms and the lateral inclination of the body. This patient has lateropulsion.

patient complains that his movements, while possible, are impeded. He finds difficulty in executing quick movements, such as turning a newspaper, steering a bicycle, etc. The rigidity of the facial muscles inhibits the quick movements that give expression to the face and hence a peculiar mask-like appearance is produced. The rigidity of the muscles moving the eyeball causes a fixed staring, and when the eyeball is moved the movement is not infrequently made up of a series of quick jerks. The rigidity of the muscles of the body and extremities results in certain characteristic positions, the fingers are usually slightly flexed at the metacarpophalangeal joints, producing the characteristic pill-rolling position, the elbows and wrists are partially flexed, and the arms adducted. The knees are bent and the body inclined forward, with a considerable anteroposterior curvature in the thoracic spine. The ulnar deviation of the fingers, to which Spiller has called attention as a mark of the resemblance of paralysis agitans to rheumatoid arthritis, may be due to other causes than the muscular rigidity.

There is usually a shuffling, slow gait with short steps. Often there is a festinated gait, *i. e.*, propulsion or retropulsion, sometimes lateropulsion, produced by the displacement of the patient's centre of gravity in a certain direction. This displacement the patient endeavors to overcome by a step in that direction, but, owing to the impediment in this motion, he is unable to regain his equilibrium and so keeps going, unable to stop until he meets with some support. This is a symptom we should expect to result from the rigidity and impediment of active motion, and it is unnecessary to bring in a hypothetical lesion of the coördinating centre to explain it, as has been done by Maillard, Zingerle, and others. There is usually no real weakness in paralysis agitans; active motion is hindered by the stiffness, but resistance to passive movement is of normal strength.

FIG. 66



This is a specimen of my hand writing

Tremor.—The tremor of paralysis agitans is highly characteristic of the disease. It is a regular, rhythmical tremor, three to five per second, constant when the part is at rest but ceasing on motion, the reverse of an intention tremor. It usually begins in the thumb, but may begin in other parts of the body or affect different parts simultaneously. When it affects the tongue, it produces coarse to-and-fro movements of that organ. It may affect the vocal cords. It is increased by excitement, disappears in sleep, and is usually absent in the period just following sleep when the mind is awake but quiescent. The head is not usually involved. When the tremor affects the fingers it frequently

produces the so-called pill-rolling movement, when it affects the feet there may be a movement produced like that of an ankle clonus. Knoblauch has reported a case of "paralysis agitans sine rigiditate," with the tremor alone the diagnostic feature, and gives a good prognosis for such cases. The accuracy of the diagnosis, however, is questionable.

Other Symptoms.—Other symptoms are of less diagnostic importance but contribute considerably to the patient's discomfort. The subjective sensory symptoms consist of feelings of heat and cold or there is an increased susceptibility to heat and cold. Not infrequently there are various forms of paresthesiæ, as of insects crawling over the skin or just beneath it. Many patients complain of the feeling of stiffness in the muscles, but sensations of fatigue resulting from the tremor are less pronounced than might be expected. Often patients feel better when riding in trains or otherwise jolted about, and advantage is taken of this in treatment. Aching pains in muscles or joints may be present, especially in the early stages of the disease. The skin may be thickened, in other cases atrophied. Local cyanosis may occur. Often there is excessive sweating. Tachycardia is sometimes a symptom. Some authors describe salivation as a symptom, but it seems probable that the increase in the amount of saliva is more apparent than real, and is due to difficulty in swallowing owing to the rigidity of the lips, tongue, and pharynx. Bulbar symptoms, such as difficulty in speech and deglutition, are sometimes reported, but they are comparatively rare.

Mental changes have also been described, but usually the patient's mind is entirely clear. In the very old the changes incident to senility, such as irritability, childishness, etc., insomnia and memory changes, might be expected and may require special treatment. Often these patients are emotionally unstable and spells of forced weeping or laughing occur. Objective sensory changes are not found in uncomplicated cases of paralysis agitans. The tendon reflexes are usually normal, but may be increased or diminished. It has been noted by Bruns, Mendel, and others that in cases of "hemiparalysis agitans" the reflexes were increased on the affected side. I examined, pathologically, a case in which the diagnosis had been hemiparalysis agitans, with symptoms of stiffness, weakness, tremor, and increased reflexes on the affected side. The necropsy showed an area of softening involving the internal capsule and the softened area filled by a concretion which had apparently irritated the motor fibers. Pathological examinations might show other cases of this kind, but they are not cases of paralysis agitans. The examination of the urine and feces usually shows an increased elimination of phosphorus, but no other abnormal constituent. The examination of the blood and spinal fluid is negative.

Differential Diagnosis.—Before beginning treatment for paralysis agitans it is necessary to carefully differentiate it from the conditions caused by senility, which it may resemble. The gait and general appearance of some senile demented persons resemble those of paralysis agitans. In senility, however, the tremor is less coarse, and frequently affects the head. When the patient has reached the period of senility it may be

impossible to say that some of the symptoms presented may not be due to this state. Hereditary or family tremors are distinguished by their constancy in the various members of the family, their non-progressive character and the absence of other signs of paralysis agitans. Tremors due to gross brain lesions are usually unilateral and there may be indications of the brain lesion in the exaggeration of reflexes; objective sensory disturbances, especially in the discrimination and localization of sensation, etc. Multiple sclerosis is a disease usually occurring at an earlier age. The tremor in multiple sclerosis is the intention type, the reverse of that in paralysis agitans, although there have been cases of paralysis agitans described as having an intention tremor. The nystagmus and scanning speech may be imitated in paralysis agitans, but careful examination will detect the difference. The Babinski reflex frequently present in multiple sclerosis is always absent in uncomplicated paralysis agitans. The staring expression of the eyes and tachycardia may cause a suspicion of Basedow's disease, which is, in fact, sometimes a complication of paralysis agitans. The tremor in Basedow's disease is very fine and muscular rigidity is absent. Rheumatoid arthritis is an occasional complication of paralysis agitans and sometimes causes difficulties in diagnosis. The stiffness in rheumatoid arthritis is in the joints rather than the muscles and the tremor is absent. Hysteria may simulate paralysis agitans very closely, but usually the hysteric is a younger individual and some of the signs of the latter trouble will be missing. If there is any doubt concerning the differential diagnosis, it can readily be eliminated by trying some suggestive therapeutics. The only form of chorea which would be likely to be confused with paralysis agitans would be the chronic progressive type, and there is but little real similarity between the irregular, incoördinate movements of chorea and the rhythmical tremor of paralysis agitans.

Treatment.—There is a general impression among physicians that, having established the diagnosis of paralysis agitans, nothing further can be done. Certainly our efforts in the past to cure this disease have met with but little result, but it would seem as though few diseases offered a better field for therapeutic research than this one. So far as we know at present there are no anatomical changes present which bar the possibility of recovery. The patient lives, and lives in hopes, and it is a well-established fact that almost any new method of treatment tried on these patients is, for a time, beneficial. Even when cure is not attempted, apparently there is much that may be done to relieve the symptoms.

Prophylactic Treatment.—No rules regarding prophylaxis can be established in the present state of our knowledge. Heredity apparently plays a minor part. Overwork, both mental and physical, plays no small part in causation, especially the continuous, unrelenting sort. The people who take their work to bed with them and who never come under the inhibiting influences of tobacco or alcohol are the kind that, according to the statistics, are most frequently affected. In this respect, the disease may be almost regarded as a badge of respectable endeavor.

Curative Treatment.—An actually curative treatment for paralysis agitans remains to be discovered. The toxic theory of Eulenberg and the resemblance of paralysis agitans to some forms of rheumatoid arthritis, as pointed out by Spiller, may be suggestions along this line. There is much in the character of the changes in the muscles to suggest that they are due to some toxic influence, though it cannot be said they are sufficiently definite to bear out the opinion of Gauthier that they are due to phosphoric acid or its compounds. Considerable experimental work has been done in the line of an organotherapy for this disease. The use of Brown-Séquard extract has not given any results. Moebius, in 1883, and Lundborg, in 1901, suggested the use of thyroid extract, but the treatment was without benefit, and Mendel has also observed no result from thyroid therapy. Parhon and Urechie, in 1908, suggested the use of extract of the hypophysis gland (two teaspoonfuls of glycerin extract, four hypophyses macerated in 30 gm. glycerin). They observed some lessening of the tremor, more quiet sleep, and improvement in the subjective symptoms. More decided results have followed the administration of extract of parathyroid gland, and there is some pathological and experimental evidence that its use in this disease would be a rational therapeutic measure. Berkley has administered the gland in 65 cases with improvement in many and apparent cure in some of them. Alquier and others have reported similar results. Dana reports improvement in about one-third of his cases by the use of this remedy, and I have seen distinct improvement, especially of the subjective symptoms, in many cases. I have seen one case in which the use of parathyroid lessened the tremor to a remarkable degree, the case becoming a typical "paralysis agitans sine agitatione." Roussey and Clunet interpreted their pathological finding in the parathyroid glands as due to a hyperplasia and suggest a radium treatment to the parathyroid region. They obtained improvement in 3 cases treated in this manner.

Symptomatic Treatment.—**MEDICINAL.**—Among the medicines used to control the symptoms of paralysis agitans the most important are probably hyoscine and duboisin. The former has been in use for many years, and, as hyoscine hydrobromate or scopolamine hydrobromate, is of great value in controlling the tremor. First suggested by Erb, the subcutaneous injection of hyoscine is recommended by Parisot, Oppenheim, Souques and Roussey, and Sataban, in doses of from $\frac{1}{2}$ to 2 mg. a day, beginning with a smaller dose and increasing the amount very gradually. The treatment may be continued for years, with good results. Good results are also obtained by administering it by the mouth, either made up or in pills, or, as I prefer, in a prescription containing also sodium bromide and chloroform water.

R—Sodii bromid.	3iv	15 5	
Hyoscine hydrobrom.	gr. $\frac{1}{10}$	0 0065	
Aquæ chloroform.	q. s. ad f 5vj	200 0	—M.

Sig.—Two teaspoonfuls three times daily.

A curative effect or even a permanent influence on the disease is not to be expected from the use of hyoscine. It is solely a symptomatic remedy, and as such exerts a good influence especially on the tremor and to some extent also on the muscular rigidity and the symptoms dependent thereon. The addition of the bromide has a tendency to lessen the nervous irritability frequently complained of by these patients. It is necessary that a good preparation of hyoscine should be used and that the solution be fresh. A few patients complain of unpleasant symptoms from hyoscine, dryness of the throat, feelings of heat, general weakness, vertigo, difficult speech, paresis of accommodation, anorexia, tachycardia, mental confusion, or stupor. Also in some cases a long use of the drug leads to symptoms on its withdrawal (heart weakness and collapse, K. Mendel).

Duboisin was suggested as a useful drug in this disease by E. Mendel, in 1893, who regarded it as the best symptomatic remedy, especially in severe cases. In none of the 12 cases in which he used it were any but good effects observed. Duboisin is less toxic than hyoscine and may be used in doses of 2 to 3 mg., two or three times daily. Francotte, Erb, and Oppenheim praise its favorable effects, especially on the tremor. K. Mendel found it very valuable in most cases but has also observed some unpleasant symptoms result from its use: In one case 0.00035 gm. duboisin injected subcutaneously produced an irregular pulse and jerking of the extremities. In another case 0.0004 gm. produced heart weakness and collapse. Mendel recommends for injection, beginning with 0.0003 gm. a day and gradually increasing the amount to 0.0005 gm.; in pills, duboisin sulphate from 0.00025 gm. to 0.0005 gm. twice a day. Hubert recommends giving an injection every second or third day and also giving it internally in the intervals.

Preparations of arsenic are also recommended by a few authors, Erb and others. For subcutaneous injections, in the form of sodium cacodylate, 0.05 gm. daily; or Fowler's solution may be given by mouth in doses of two to four drops, three times daily. Patients receiving arsenic usually feel better and stronger but there seems to be no effect on the rigidity or tremor. Oppenheim recommends tincture veratrum viride in doses of from three to five drops, three times daily, for the relief of the tremor. Tincture gelsemium is also recommended. Cannabis indica in pills of $\frac{1}{4}$ gr. each, t. i. d., may be of service, according to Starr. Atropine has been recommended by Hansen, Eulenburg, and Verhoogen, according to K. Mendel, and he indorses a prescription of Rabow:

R—Atropin. sulph.	gr. iij	0/2
Ergotin.	gr. xxx	2/0—M.
Ft. pil. no. xxx.		
Sig.—One pill three times daily.		

He also recommends:

R—Potass. iodid.	3ss	6/0
Ergotin.	gr. xv	1/0
Aquæ dest.	f 5vj	200/0—M.
Sig.—One teaspoonful three times daily.		
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On account of its apparent relation to chronic rheumatic troubles the use of salicylic acid or its salts might be suggested, especially for the relief of the pains and paresthesiæ that are sometimes present, but few authorities recommend them, however, and Oppenheim speaks of having seen harm result from their use. Quinine and the mineral acids are recommended by Dana for the relief of vasomotor and sensory symptoms. Codeine and morphine, although recommended by some, should certainly never be used except in the last stages, both because of the danger of the habit formation and the unfavorable effect on the general health of the patient. Veronal may be given occasionally for insomnia, but, as a remedy to be given daily, is, according to K. Mendel, without effect.

HYDROTHERAPY.—There seems to be considerable difference of opinion as to the value of baths in this condition and also as to the kind of hydrotherapy which should be used. Oppenheim advises strongly against any extreme measures, but a mild rubbing with lukewarm water or a cool half-bath may have a good effect, and Erb is of the same opinion. Wollenberg advises prolonged warm baths followed by a cool rub. Dana says that hot baths "are agreeable and helpful." While Starr favors a twenty-minute bath in tepid water not followed by cold shocks. It is generally agreed that cold baths are harmful and this applies also to ordinary sea-bathing. The bath-cure establishments rarely benefit these cases. Hot baths, mud baths, alkaline baths rarely are beneficial and are frequently harmful. Each case must be treated according to the symptoms present. The warm baths are beneficial for those cases complaining of pain and marked rigidity. A washing with cool or tepid water is of benefit for the paresthesiæ and is more beneficial to the general health of the patient.

MASSAGE.—Light massage, either general or with special attention to the parts affected, is of considerable benefit in lessening the rigidity. Starr advises especially the lomi lomi method as practised in Hawaii. Great care must be exercised as too strenuous or prolonged manipulation may do harm. Even massage, as ordinarily given by trained masseurs is, in many cases, productive of unpleasant results. On the other hand, rubbing or stroking movements combined with gentle kneading have at least the effect of lessening the rigidity and making the patient feel better. Massage with the mechanical vibrator has no advantages over hand-massage and when used it should be with the lightest pressure and with soft rubber applicators.

Vibratory massage applied to the spine, as advised by the makers of these machines, has not been productive of any permanently beneficial results in my hands. The use of the Zander apparatus by which large vibrations can be communicated to the patient's body has a temporarily beneficial effect on the tremor. The observations of Charcot and others that patients sometimes felt better after riding in trains, etc., led to the treatment of the condition by seating the patient on a chair which could be mechanically shaken, the "*fauteuil trepidant*." No permanent improvement is obtained by this method and the treat-

ment is now abandoned. Glorieux advised two of his patients to ride one hour daily on an omnibus and claimed that they felt better, walked with greater ease and elasticity and had less tremor, but the improvement was of short duration. Suspension treatment is a method in little use at the present day, but Charcot and de la Tourette claim that in 4 cases treated by this method there was improvement in sleeping, also the paresthesiæ, muscular rigidity, and pain were lessened. Eulenburg and Mendel found slight improvement in one case but none in another. Nerve-stretching gives no improvement (Oppenheim).

ELECTRICITY.—The results obtained by electrical treatment will depend to a great extent upon the knowledge and skill of the person giving the treatment, and this is true of paralysis agitans as of other diseases. The bipolar faradic bath is recommended by Oppenheim and by K. Mendel. The mild galvanic bath is also useful. Toby Cohen recommends the cathodal baths. The results obtained are only temporary. The current strength should be moderate, duration ten to fifteen minutes, and treatment should be given three times a week. Various ways of using the galvanic current have been suggested. Galvanization of the head and neck in early cases leads to a lessening of the tremor. Anodal galvanization of the hypertonic muscles leads to some lessening of the rigidity. Ingria found temporary improvement from placing the anode on the nape of the neck and the cathode on the last cervical vertebra. Verhoogen, to relieve the muscular rigidity, used the anode of the galvanic current on the rigid muscle and treated the antagonists with the faradic. In my hands the best results have come from using the galvanic current, placing a large cathode at some indifferent point and moving the anode slowly over the rigid muscles, using a fairly strong current, but being careful not to have any sudden changes in the current strength. Treatments should be given daily. The faradic current is of no use in this affection, and may increase the symptoms. The sinusoidal and high frequency currents have not had sufficient trial to be sure of the results. They have a sedative effect in some cases.

EXERCISE.—A detailed description of the use of exercise in paralysis agitans is given by Friedlander, and Roth and Laserow have used his methods with good results. He claims that even in severe cases of long standing the condition can be improved and the progress of the disease halted, while in the early stages there is a "decided improvement." The treatment at first consists solely of passive movements of all the joints of the parts affected, individually and slowly, the movements at first to be of small degree and each joint flexed and extended five to ten times at each seance. During the exercises the patient endeavors to relax the part as much as possible. This is followed by active exercises directed chiefly to the extensors, the theory being that an active voluntary movement of the extensor muscles will lead to a corresponding relaxation or inhibition of the flexors and hence overcome the flexor contracture. This theory is based on the physiological teaching of Sherrington, Hering, and others that the innervation of an agonist

muscle is accompanied by an inhibitory influence on its antagonist. Special attention should be paid to standing and walking movements, in order to correct the deformity of the spine; and special exercises are employed for the fingers in order to improve the handwriting. Friedländer lays stress on his "falling exercise," in which a certain part of the body is passively raised and then allowed to fall; the patient endeavoring to keep the part entirely relaxed at all times. He believes that by this exercise the patient's power of voluntary relaxation is much increased. Care should be taken in giving any of these exercises that the patient is not tired by them. They should be done regularly and every day or several times a day.

CLIMATE.—Climate exercises but little influence on the course of the disease, but most of these patients feel better in a dry, warm climate, and warmth also has a favorable influence on the rigidity. Oppenheim favors wooded and low mountain regions. Nolda believes that high mountain regions have a favorable effect (St. Moritz in the Engadin). Certainly the atmospheric conditions should be such that the patient can have plenty of fresh air without being exposed to cold or wet.

PSYCHOTHERAPY.—Hypnotism is said to have no effect on this condition. Educational exercises such as were suggested by Brissaud for the treatment of tics of psychic origin have very little, if any, effect on the general symptoms, although the handwriting may be a little improved in this way. It has been mentioned that almost any new remedy for the condition causes some apparent improvement at first so that the physician should be prepared with many remedies, not to use together, but that he may use them one after another and continue to inspire some hopefulness in the patient during the course of this prolonged disease.

General Considerations.—As these patients are usually mentally alert, idleness is likely to be a great hardship, therefore, it is better to allow a patient to continue his occupation unless it is one that involves considerable excitement and mental strain. Even in advanced cases they should be kept occupied with some non-exciting light labor, such as gardening, etc. These patients are best kept free from all exciting influences, hence journeys to strange places, bath-cure resorts, etc., often do more harm than good. The diet should be light and easily digested. I have had patients who felt better when on a purin-free diet, and it is usually advisable to restrict the use of meat to small quantities. Alcohol and tobacco in moderate quantities apparently cause no bad effects. The sleeping hours should be moderately long and the patient may be encouraged to remain in bed for a time after waking in the morning and also to lie down frequently during the day for short naps. Regulation of the bowels is an important factor in the comfort of the patient, and it is also important to bear in mind that at the age when this disease is most prevalent there is a tendency to enlarged prostate. The urinary retention and frequency of urination due to this may be a source of irritation to the patient and aggravate the symptoms. Brown reports a case of hereditary tremor in a man,

aged thirty-four, which was cured by the removal of an enlarged cystic middle turbinate and polyp and cauterization of the stump. There was a very distinct family history of tremor of "head and hands" from the paternal side. The patient's symptoms, consisting only of tremor of the head, disappeared after the first operation, reappeared in about four months, but again disappeared after a second cauterization. The case was not paralysis agitans, but serves to show that local sources of irritation may increase the nervous irritability of these patients and that all such conditions should be examined for, and removed, if necessary, by surgical intervention.

MULTIPLE SCLEROSIS

Multiple sclerosis (disseminated sclerosis; *sclerose en plaque*; sclerosis multiplex cerebros spinalis) is a disease of great interest to the physician because of the great variations in the clinical picture of the disease and the frequency with which it is mistaken for other conditions. Its therapeutics also opens up a wide field of investigation. The long remissions and the change in symptoms from time to time seem to point to an essentially curable condition if the proper measures were employed.

While the incidence of the disease is probably greater between the ages of twenty and thirty, it is not very unusual for it to appear earlier in life. Eichorst reported a case with necropsy in an eight months' old child born of a mother with the same affection. Very seldom does the disease develop later than the age of fifty, but, as the disease runs a very slow course, it is not uncommon to find it present in old people. I have seen many patients past sixty years of age with this disease and in almost all of them the onset of the affection dated back twenty to forty years.

Opinions differ as to its frequency. Some of the best observers in continental Europe (Oppenheim and others) believe it to be one of the most frequent organic diseases of the nervous system. American writers usually regard the disease as rare. Starr, for instance, reports 109 cases of multiple sclerosis in 31,502 patients. In 1905 Spiller and Camp, reporting two cases with necropsy, could find only six cases in the American literature with necropsy, but since that time a number have been reported. Certainly one factor in its supposed rarity in America is the difficulty in diagnosis. Some observers regard it as being more frequent in males. My own experience has been that the sexes are about equally affected.

Etiology.—The etiology of the disease is uncertain. Strümpell holds that the cause lies in some factor inherent in the individual's nervous tissues, a theory which is not generally accepted. It frequently follows some infectious disease—typhoid fever; scarlet fever; measles; influenza; cholera; whooping cough; and acute rheumatism (Oppenheim); malaria (Spiller); diphtheria (Henschen); the puerperium (Hoesslin). It has also been attributed to zinc poisoning (Schlockow), manganese

poisoning, mercury poisoning (Gerhardt), and poisoning with *aspergillus fumigatus* (Ceni and Besta). Syphilis is not a cause of multiple sclerosis, although a positive Wassermann has been found in apparently non-syphilitic cases. Trauma, especially if combined with worry or mental shock, has been regarded as a cause in some cases. Spiller and others have called attention to the occurrence of the disease after exposure to cold, and a history of such exposure has been very frequently obtained in my cases. It is quite possible that the cold acts by lowering the resistance to some infection, in much the same way as exposure to cold is a cause of pneumonia. Also, it seems very possible that the acute infections above mentioned act by lowering the resistance of the individual to some special infection.

Pathological Anatomy.—The pathological anatomy consists of sharply defined areas of sclerosis scattered through the central nervous system, often affecting one part more than another, sometimes affecting only certain parts. It is this variability in the distribution of the sclerotic foci that gives rise to such great variations in the clinical picture of the disease. With the unaided eye and under low powers with the microscope, the patches appear to have sharply defined margins and even with the higher powers, the margins are more definite than areas of softening caused by cerebral thrombosis. They affect both white and gray matter, show no tendency to involve any special tracts of the spinal cord, and vary greatly in size, some being so small as only to be visible microscopically. Within the sclerotic areas the myelin sheaths of the nerve fibers are degenerated and their place is taken by a growth of neuroglia, but the nerve fibers themselves, the axis-cylinders, usually persist, as can be shown by special staining methods. The fact that many of the axis-cylinders persist in the sclerotic areas explains why the secondary degeneration from those areas is comparatively slight. While traversing these patches the axis-cylinder is without its sheath, and is, therefore, uninsulated from others in the vicinity. It is possible that this condition gives rise to a confusion of the impulses being carried over these fibers and that some of the symptoms, such as intention tremor, etc., are produced in this way.

Siemerling and Raecke have made very careful studies of these patches and their conclusions are of great importance. They found that the sclerotic areas were always in relation to bloodvessels, and that especially in the smaller and more recent areas this relation was pronounced. In these foci there was a round-cell infiltration about the bloodvessel, which in the older plaques was absent. This cellular infiltration had also been noted by Oppenheim and by Spielmeyer and means much in any consideration of the pathogenesis of the condition. In the fibrillar preparations, by the Bielschowsky method, there was invariably, in the more recent foci, an area in which the fibrillæ appeared disintegrated. The middle point of this area was a bloodvessel, or, if no bloodvessel, some blood pigment. These small hemorrhages have also been observed by others—Strahuber, Schuster and Bielschowsky. Siemerling and Raecke conclude that the develop-

ment of the plaques in multiple sclerosis is due to an extension of small myelitic or encephalitic foci in connection with the bloodvessels. It is an inflammatory process, proceeding from the region of the bloodvessel. First there is a small hemorrhage with slight involvement of the neurofibrillæ and more marked destruction of the myelin sheath. The glia proliferation is partly a reaction to an irritant and partly a simple formation of scar tissue. The condition might be caused by an intoxication or poison or by a chronic infection.

Symptoms.—The classical symptoms of nystagmus, scanning speech, and intention tremor cannot be relied upon to make a diagnosis of this condition. The nystagmus is present only in from 50 per cent. (Frankl-Hochwart, Schultz) to 75 per cent. (Müller, Marie) of cases of multiple sclerosis, and it is present in many other conditions, such as cerebellar affections, labyrinthine conditions, etc. Scanning speech was present in only 15 per cent. of the cases in Müller's own clinic, in only 25 per cent. of the cases he collected from the literature. The intention tremor is a more common symptom, but even it was absent in 30 per cent. of Müller's cases, controlled by autopsy findings. The comparative rarity of this disease in some American clinics may be explained by the fact that some observers rely on the above symptoms to diagnosticate the disease. While the presence of the above triad of symptoms permits the diagnosis to be made, there are usually other symptoms of equal diagnostic importance and symptoms which require that measures be taken for their relief.

Among the most common symptoms complained of by these patients is headache, which was present in one-third of Müller's cases and in practically all of mine. It is sometimes severe and continuous and, when accompanied by vertigo and vomiting attacks, may give rise to suspicion of brain tumor. Sometimes when coming on in attacks it is diagnosticated as migraine. Often it is a diffuse, pressing pain, not unlike that called neurasthenic. Attacks of vertigo are not uncommon. True apoplectic attacks are very rare, but apoplectiform attacks, transient loss of consciousness, followed by a transient weakness or transient sensory changes are not at all unusual. Epileptiform attacks may also occur. Attacks of the Jacksonian type of epilepsy have been described by Gussenbar.

The mental state of many of these patients is profoundly influenced, though it has little that is characteristic. Excitement and deterioration are rare, although Oppenheim speaks of "transient dementia and confusional attacks." Persecutory ideas are frequently found, having been described as a characteristic by Dannenberger, Charcot, Bruns-Stolting, and others. The most frequent mental state is a simple depression which is sometimes pronounced and accompanied by suicidal tendencies. On the border-line, between a neurological and psychological manifestation, is the forced laughing and crying, seen in more than half of these patients. Frequently it is unaccompanied by any emotion, and is supposed to be due to lesions in the basal ganglia.

Ocular and visual symptoms are very common. The frequency

of nystagmus has been mentioned. Transient ocular palsies occur in about 40 per cent. of the cases, causing transient diplopia. A loss of pupillary reflexes has been described and also Argyll-Robertson pupil. The finding of the Argyll-Robertson pupil should lead to very careful investigation for a possible syphilitic origin. Changes in the eye-ground appearance are found in over 50 per cent. of cases, according to Uhthoff, and a pallor of the temporal side of the optic nerve head is most characteristic of the disease. Optic neuritis and choked disk rarely occur. Various changes in vision have been described.

It is characteristic that there is usually an apparent disproportion between the fundus changes and the visual disturbance. Total amaurosis is rare. More commonly there are defects in color vision or in the visual fields. Klingmann has described the frequent occurrence of multiple scotomata in the visual fields.

The typical scanning speech, when it is present, is highly diagnostic, but other changes in speech may occur. A spastic paresis of the pharynx may lead to symptoms resembling bulbar palsy, and this may be combined with some difficulty in swallowing. Salivation is not a symptom of multiple sclerosis.

Respiration and heart action are not usually interfered with, although Müller has reported a case accompanied by attacks of paroxysmal tachycardia in which it was possible that a sclerotic patch in the medulla might have been responsible for the heart symptoms. Paralytic symptoms in the face and tongue occur but rarely. Trigeminal neuralgia is not infrequent and, according to Oppenheim, may be one of the first symptoms of the disease. The same author is authority for the statement that a nerve deafness is sometimes a symptom.

FIG. 67

This is a specimen of my handwriting

The motor symptoms are very important not only as diagnostic phenomena, but also because they are, in many cases, the symptoms of which the patient most complains. In the early stages of the disease, ataxia of the arms or legs may be present and precede the development of any other motor disturbance. This ataxia is rarely accompanied by changes in sensation in the part affected, as is the case in tabes dorsalis, polyneuritis, etc. The gait is of a staggering, irregular type, not markedly affected by closing the eyes, and accompanying this

there is usually some stiffness in the legs, giving a spastic element. The irregularity of the gait may be further increased by an intention tremor in the legs. The intention tremor is usually most pronounced in the hands, showing itself in attempts to convey food to the mouth, in the handwriting, etc.

The tendon reflexes are usually increased unless, as in a case reported by Spiller, a sclerotic patch lies directly in the path of one of the reflexes, in which case that reflex would be lost. The skin reflexes, on the other hand, such as the abdominal and cremasteric, are usually lost. This dissociation of reflexes, increased tendon reflexes and lost skin reflexes, is very characteristic of the disease. The Babinski reflex is present in a large percentage of cases of multiple sclerosis and its presence is of great value in the diagnosis of this disease from hysteria. Muscular atrophy is rare but is sometimes present in this affection.

The sensory changes of multiple sclerosis have been repeatedly investigated. One of their chief characteristics is their fluctuating character. They occur in more than two-thirds of all cases and are both subjective and objective in character. Severe pain may be for years the only symptom of the disease (Gebhardt). It is only rarely of typically neuralgic or lancinating character, more frequently it is a dull ache, sometimes in muscles, sometimes in joints, but usually moving about and with marked variations in intensity. Paresthesias of various kinds may be present. They usually are more marked in the distal portions of the extremities. Objective sensory disturbances are frequently found and may affect different forms of sensation and be of various distribution. A hemianesthesia is sometimes present; at other times there are patches of anesthesia.

Disturbances of the function of the bladder occur in at least three-quarters of the cases. Often this takes the form of transient attacks of retention. Rarely does the symptom become permanent. Müller describes as a frequent symptom in the early stages a peculiar, dull sensation in the bladder region accompanied by difficulty in starting the flow of urine. Some disturbance of rectal function is found in many cases. It is usually of the same character as the bladder disturbance, but the obstipation may alternate with attacks of diarrhea. Menstrual irregularities occur not infrequently. Vasomotor and secretory symptoms are not uncommon—hyperidrosis and local flushings, etc. Sometimes the disease is accompanied by one of the various angioneuroses, such as erythromelalgia (Collier, 5 cases; Cassierer, 1 case). Examinations of the blood, urine, and cerebrospinal fluid are usually negative.

Many atypical cases of multiple sclerosis occur and attempts to classify these cases have been made by Oppenheim and others. There is little to be gained by a clinical classification of types of the disease, since the symptoms depend entirely on the number and location of the sclerotic patches, thus permitting of almost infinite variations.

Differential Diagnosis.—Multiple sclerosis may resemble, to a greater or less extent, a great many other diseases of the nervous system;

the differential diagnosis in some cases being exceedingly difficult and only possible by the most painstaking examination. Many of these cases are diagnosticated as neurasthenia and hysteria, but Babinski's phenomenon, fundus changes, etc., do not occur in these affections. Paralysis agitans is characterized by a tremor at rest; multiple sclerosis, by an intention tremor. The other symptoms of multiple sclerosis are absent in paralysis agitans. Apoplectiform attacks and epileptiform attacks may lead to the diagnosis of apoplexy or epilepsy, and in both cases the other symptoms of multiple sclerosis will be necessary to establish the diagnosis. Cerebrospinal syphilis and the metasymphilitic diseases, such as tabes and paresis, may be readily confounded with multiple sclerosis, especially if they present an atypical clinical picture; perhaps the most reliable method of differentiation is the chemical and cytological examination of the spinal fluid and the presence of the positive Wassermann reaction in the spinal fluid (using 0.6 c.c. of fluid).

The symptoms of headache, vomiting, and vertigo occurring in a case of multiple sclerosis may suggest the diagnosis of brain tumor especially if some optic neuritis is found. Brain tumors, however, if in the cerebrum, may produce definite localizing signs, but are not accompanied by the other signs of multiple sclerosis; if in the cerebellum, the gait, speech, nystagmus, etc., may resemble closely the signs of multiple sclerosis, but in such cases a high degree of choked disk is usually an early symptom. It must be remembered that some cysts of the cerebellum and tuberculomata may be very difficult to differentiate from multiple sclerosis. Some cases of acquired hydrocephalus, through the pressure on the cerebellum, produce symptoms which may be mistaken for those of multiple sclerosis, but the diagnosis in this event is usually not difficult.

Amyotrophic lateral sclerosis, especially in the early stages, may cause confusion, but intention tremor, nystagmus, scanning speech, etc., are absent in this affection. Acute disseminated encephalitis and encephalomyelitis may produce symptoms indistinguishable from multiple sclerosis, but the course of these conditions is not the same. Family spastic paralysis and the hereditary cerebellar ataxias may be distinguished by their hereditary character and the absence of some of the symptoms of multiple sclerosis, especially those in the sensory sphere. Cerebral arteriosclerosis is not usually difficult of diagnosis; the age of the patient and the blood pressure, the sclerosed peripheral arteries and the permanency of the symptoms are good diagnostic criteria. Diffuse sclerosis, a condition in which there is a diffuse overgrowth of neuroglia throughout the central nervous system, does not give the symptoms of multiple sclerosis in a characteristic form and is usually accompanied by dementia. Pseudosclerosis (Westphal-Strümpell), a condition in which there are no anatomical changes discovered in the central nervous system, has symptoms similar to those of diffuse sclerosis. The diagnosis from a diffuse sclerosis is frequently impossible.

The symptoms of chronic manganese poisoning and chronic mercury poisoning may resemble multiple sclerosis very closely; the history, and the relief of the symptoms by removal of the cause, are sometimes necessary to establish the diagnosis of these conditions.

Treatment.—Since the development of the idea of an exogenous origin for the affection, such as some toxin or infection, there is reason to hope that an efficient prophylaxis may some time be discovered. At present, however, we can do nothing in this direction.

The disease is usually of long duration and not in itself fatal. Spontaneous remissions frequently occur, of greater or less duration. Oppenheim speaks of having seen six cases in which remissions had occurred in which, at the time of writing, there had been a period of ten years of freedom from symptoms. Cases in which the diagnosis has been established with clinical certainty are very rarely, if ever, cured. P. Marie has suggested that an antitoxin might be discovered for the disease, but nothing has been accomplished in that direction. The observation of Oppenheim that in a case of multiple sclerosis all evidence of the disease disappeared following an attack of facial erysipelas is interesting in this connection.

Buzzard calls attention to some analogies between multiple sclerosis and cerebrospinal syphilis, and suggests that the former is due to some parasite similar to the treponema, which further study may be able to isolate. It is well known that mercury has no beneficial effect in multiple sclerosis, and Buzzard therefore suggests that arsenic be used as an internal antiseptic, either the salvarsan injection or in other combinations. He does not report any experience with this treatment. Kleineberger reports having given salvarsan intravenously to three patients with multiple sclerosis without beneficial result. Arsenic in various forms has been used a great deal in the treatment of this disease, and, in my experience, has given by far the best results of any form of medication. My practice has been to give it either in pills of arsenous acid or as Fowler's solution, so that the dose may be gradually increased to the limit of tolerance and then kept just below this point for about two weeks. After an intermission of about two weeks the treatment is repeated. Whether arsenic administered in this manner acts in the way suggested by Buzzard or has only a general tonic effect is not determined by the result. A very marked improvement may occur, but often the patient relapses. Another method of administration is by the subcutaneous administration of sodium cacodylate. Frequently good effects are obtained by giving arsenic in combination with various tonic medicines, such as the elixir ferri quinine et strychnine, U. S. P., or as in a prescription recommended by Müller:

R—Acid. arsenic.	gr. $\frac{1}{2}$	0'05
Extr. strych.	gr. viij	0'5
Quin. hydrochlor.,		
Ferri lacti	aa gr. viij	0'5
Extr. gent.		q. s.—M.
Ft. pil. no. c.		
Sig.—One or two pills three times daily.		

The relation of some cases to malarial infection suggests the use of quinine in any case in which such an origin may be suspected.

Mercurial treatment is rarely beneficial in multiple sclerosis, but on account of the impossibility of differentiating, clinically, some cases of multiple cerebrospinal syphilitic lesions from multiple sclerosis, in the absence of the data to be obtained from lumbar puncture and the Wassermann reaction, it may be advisable to try the effects of mercurial treatment for a short time. Muhsam and Dana both speak of having obtained good results from the use of antisyphilitic treatment in cases diagnosed as multiple sclerosis. Oppenheim speaks of having seen an optic neuritis develop in the course of an "inunction cure," but it rapidly subsided. The administration of potassium iodide is not usually followed by any beneficial results.

Silver nitrate is a remedy frequently recommended and highly praised by some, especially older writers. In more recent times its use is more or less abandoned. Oppenheim reports having used the unguentum Credé in a number of cases, and thought that in a few of them it was followed by good results.

Salicylic acid and its salts, also salicin, aspirin, etc., have been recommended as curative medicines, but their effects seem very temporary. Starr suggests that cod-liver oil is of service in these cases, and Dana records a case in which a remission occurred while the patient was receiving phosphates. Chloride of gold, ergot, iron, physostigmine, and zinc are the other remedies which have, from time to time, been suggested and received support from some observers. The occurrence of spontaneous remissions probably accounts for the belief in the helpfulness of so many remedies when there is no valid reason why they should have any action whatever on the disease.

Oppenheim recommends a diaphoretic treatment in those cases in which the course of the disease takes the form of acute attacks. A rest treatment is of value in some cases, more especially in those in which fatigue, weakness, and loss in weight are prominent symptoms. Even without the complete rest treatment much benefit may be derived from keeping the patient as quiet as possible, forbidding all excitement and special exertion and insisting on regularity in meals, etc. Many observers have noted the beneficial effects of visits to cure resorts, such as Nauheim, Oeynhausen, and others. While hydrotherapy is not of much value, tepid baths or salt baths at 95° F., as recommended by Starr, may be of some benefit. Very hot or cold baths are to be avoided. Electrical treatment is not usually followed by any marked improvement. Oppenheim recommends galvanization of the head and back. The violet rays have been used with apparent benefit (Dana). General massage and passive movements in the more advanced cases, or moderate exercise in fixed amount and preferably in the open air in the milder cases, are measures of value in keeping up the patient's general nutrition and should not be neglected.

Symptomatic Treatment.—In all chronic diseases one of the most important features of any treatment is the securing of relative comfort

to the patient by the amelioration of those symptoms which most annoy or distress him. The neuralgic pains and headaches may be combated with the coal-tar products, acetphenetidin, gr. v to x, pyramidon, acetanilide, etc., although care should be taken that the patient takes them only by the advice of the attending physician. Salicylates, and especially aspirin and salicin, are frequently of value in relieving pain. Local application of heat is sometimes effective, or a light, stroking massage of the part. The local application of the galvanic current may be used for the same purpose, the anode being applied to the painful area and a 2 to 5 ma. current allowed to pass for from five to fifteen minutes. The electrode should be slowly moved about over the skin and care should be taken to avoid any shock by suddenly making or breaking the current. A lumbar puncture had a markedly beneficial effect on the pain in one of my cases, even though there was no increase in the intracranial pressure and no abnormality in the fluid; the patient remained entirely free from pain for over a week.

The possibility of attacks of vertigo, faintness, syncope, etc., should be borne in mind and the patient warned against going into dangerous situations. Dana recommends the administration of atropine or belladonna for the vertiginous symptoms. Usually the vertiginous attacks result from rapid change of position, and may, with care, be avoided.

The mental symptoms are sometimes very much in evidence and increase greatly the difficulties of the care of these patients. When there are paranoid tendencies the attitude of opposition to treatment and suspicion of the good intentions of those caring for the patient frequently result in such a complete disregard of orders that successful treatment can only be carried out under restraint in an institution. The simple mental depression which is more commonly present may be one of the chief complaints of the patient. Something can be done by providing cheerful and congenial companionship, or, if the patient is able, an interesting occupation. The occupation that might prove useful in this way would have to be selected to suit the individual case, and care should be taken that the success of the enterprise is not defeated or rendered difficult by the patient's physical infirmities else the patient becomes discouraged and more depressed than ever. Suicidal tendencies are not uncommon in multiple sclerosis, and two of my patients have actually committed suicide, one by hanging, another by leaping from a window. In both cases the act was apparently impulsive, since no special previous preparation, such as writing farewell letters or adjusting financial affairs had been made, and in neither of these cases had the tendency been very apparent. Psychotherapy is not effective in relieving the mental state aside from the temporary effect of reassurance and suggestion. Hysterical phenomena complicating the case may be relieved in this way.

The paralysis is usually of the spastic type. The spasticity may be relieved temporarily by warm baths, light massage, and passive movements. The relief usually does not last more than an hour, and is not

worth while unless the patient complains of the feeling of stiffness. Owing to the fluctuating character of the symptoms, surgical operations for the relief of spasticity, such as Förster's operation, the resection of the posterior spinal nerve roots, are not to be recommended. Electrical stimulation to the paralyzed muscles seems to have no good effect, and, if anything, increases the spasticity.

The treatment of the staggering depends upon the mechanism of its cause, which is probably totally different in different cases. When the staggering depends upon an interference by a plaque of sclerosis, in the path of coördinate motor impulses no treatment will be of value save the removal of the plaque, and the same is true if the equilibrium is destroyed by a lesion of the equilibratory centre; but if the trouble consists of an interference with sensory impulses, muscle and joint sensibility from the lower extremities, then, by educating another sense to take their place, the defect can be annulled. It is in the latter case, which may be diagnosticated by sensory examination of the lower limbs, that the Frenkel reëducational exercises may be expected to be of benefit. No exercise should be taken that causes any unpleasant sense of fatigue.

The tremor is distinctly less when the patient is mentally at ease, and therefore the surroundings of the patient have much to do with it. Occasions when the tremor is likely to be particularly annoying, such as in writing or at table, should be surrounded with precautions against excitement. Veronal has been recommended by Oppenheim as a useful drug to control the tremor. It probably acts only by lessening mental excitement.

Retention of urine may require special treatment. It is usually a transient symptom and may consist simply of difficulty in starting the stream. Simple measures, such as the sound of running water, local application of heat to the lower abdomen, or pressure on the abdomen, may be sufficient to give relief, but the catheter may be necessary. As the retention is usually due to an increased reflex activity of the bladder sphincter, any source of local irritation should be cared for and the urine rendered as unirritating as possible by drinking large quantities of water.

The visual disturbances are not usually much benefited by treatment directed specifically to them. Vibratory massage to the eyeballs appears to be of some benefit in those cases where a distinct optic atrophy exists. Uhthoff has called attention to the fact that the vision is improved after prolonged rest of the eyes, especially rest in a darkened room.

Diet does not seem to influence the disease to any great extent, so that a general mixed diet consisting of such articles as are easily digested may be recommended. I have used a purin-free diet in several cases and believe that it was beneficial. A warm and dry climate is of benefit for the pain, but in other respects the climate does not seem to affect these patients.

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CHAPTER XVI

THE TREATMENT OF THE TOXEMIAS OF DANGEROUS TRADES AND OF DRUGS

By LOUIS CASAMAJOR, M.D.

DANGEROUS TRADES: METALLIC SUBSTANCES

General Prophylaxis.—It was to the metallic substances, primarily lead, that interest was first attracted when the study of trade diseases was first inaugurated. Men had known for years that certain trades bestowed disease and death upon workers, but it was only after the birth of the idea of employers' responsibility that organized government began to take cognizance of this fact. It was soon seen that most of these disablements were preventable and that proper precautionary measures would conserve the health of the workmen—a real asset to the State.

Prophylaxis then must be the main element in the handling of trade diseases, even more than in diseases of other etiology. This phase of the situation is, in the main, a legislative one, and deserves only passing mention in a work on medical treatment.

In the civilized countries of the present day, there are laws being constantly enacted to protect the workers in dangerous materials, and most of these have to do with improving working conditions and thus minimizing the danger of contact with poisons. As the prophylaxis of all mineral poisons has points in common, this subject may be considered for all together. First, the mineral poisons handled in industry. The most important of these, both from the point of frequency and danger, is lead. After this come arsenic, mercury, copper, and manganese.

"Dust and fume," says Oliver, "are the greatest foes of industrial life," as it is by these means that most of the industrial poisonings occur. The dust in the air is taken into the mouth, the air passages, and lungs, and, most important of all, swallowed with the saliva. Probably none of the metallic poisonings occur from absorption through the lungs. The pulmonary epithelium is occupied solely with the absorption of gases, and whatever solid matter enters the lungs is probably never allowed to enter the general circulation. When a small amount of dust enters the lungs, as in the case of every city dweller, the phagocytic blood cells take it up and dispose of it, usually by storing it in the lymph nodes at the bases of the lungs and in the interstitial tissues. When large amounts of solid matter are inspired, much of it is disposed of in this manner, while the remainder is taken up by the phagocytes,

derived both from the blood and from the lung parenchyma itself. Later this finds its way into the bronchi and is coughed up, giving the dark colored sputum which most dust workers show.

When the poison is swallowed, the procedure is different and industrial poisons are swallowed in the first place in the form of dust, which attaches itself to the moist mucous membranes of the mouth and nose and passes down to the stomach, with the swallowed saliva and nasopharyngeal mucus. Secondly, workers who are not careful as to the condition in which their food and hands are kept, swallow considerable quantities of the materials they work with whenever they take their meals. Every worker who eats with the dust of toil on his hands takes in a measurable quantity of the poison with every bit of food he handles. In other trades, such as typesetters and flower workers, where lead products are held in the mouth, it is the dissolved and swallowed poison which is responsible for the toxemia.

When the metallic substances reach the stomach they find there a warm acid medium which favors solution. All of the metals mentioned have soluble chlorides and any number of soluble albuminates and other protein products. Rambousek has shown that the solubility of metallic lead and white lead is much increased by the presence of 1 per cent. of peptone. Absorption of the heavy metals takes place in the intestines. While the greatest percentage of these substances pass through with the feces, nevertheless some of the matter is absorbed by the healthy epithelial cells; probably more when ulcers exist, due to local action of the poisons. When the salts of the metals enter the circulation, they are taken up and stored by practically all parts of the body, but principally by the liver and kidneys. While absorption is a slow process, excretion is a much slower one, so that with continued poisoning, such as the workers are subjected to, there is a cumulative action of the ingested poison. Excretion takes place for the most part through the lower bowel and to a lesser extent through the kidneys, although traces of lead have been demonstrated in the milk and saliva. It is a matter of grave doubt whether the skin glands participate in the excretion to any extent.

Importance of Cleanliness.—From this short summary, it becomes obvious that the prophylaxis of the mineral industrial poisons may be summed up in the word "cleanliness." Cleanliness first in the air the workers must breathe. This must be brought about by the elimination of dust and fume. This may be accomplished, when practicable, by changes in the method of handling materials so that finely pulverized matter is not handled at all, or handled in the wet state, in which no dust can arise. When this is impracticable, proper ventilation must be brought about in the workroom. Large airy rooms minimize the danger by lessening the concentration of dust in the atmosphere. Forced draft ventilators and the installation of exhaust shields over furnaces, machines, and work tables do much to lessen the danger. When it is possible, work in the open air instead of under cover frequently solves the problem.

More difficult than this is the question of personal cleanliness of the workers. Here one meets an individual problem of extreme difficulty. Familiarity ever breeds contempt, and it is no easy matter to convince workers, especially those of limited intelligence, that danger lurks in this element of their every-day life. All those who have attempted to force workers in dust to use air filters which are attached over the mouth and nose will testify to the uselessness of these contrivances. Not that they are in themselves useless, but it is impossible to induce workers to use them. They are clumsy, uncomfortable things at best and interfere with talking, and the workers believe, breathing, and are soon discarded. The author tried the experiment of having some men who work in thick dust supplied with these filters. After a week, no man was using them and those who still carried them in deference to the rules usually wore them hanging from their belts.

The question of inducing workers to wash the hands before each meal presents the same difficulty, especially among those who from childhood up have believed that washing is rather a luxury of the effete than a matter of necessity. Nevertheless, every workshop where dangerous substances are handled should be equipped with a proper washroom, with hot water and plenty of soap and towels, and the workmen urged to utilize these conveniences. At any rate, an absolute prohibition can be placed on workmen eating at the place of work or near the materials of their trade. A final word must be said here upon the practice of tobacco chewing as a prophylactic agent, which amounts practically to a superstition among certain metal-workers, notably those in brass and zinc. This is undoubtedly more than a superstition, for when we reflect that most of the poison is swallowed with the saliva, we readily see how the tobacco chewer with his continual spitting would swallow much less during the day than would his non-chewing fellow-worker. In the industrial toxemias, as in everything else, prophylaxis is the important thing, for the treatment of the severe conditions when they occur is not happy in its results.

Lead.—Incidence.—Lead is undoubtedly responsible for more of the industrial toxemias than any other substances, as it is one of the most extensive in use. Not only is metallic lead used both in the pure state and in alloys with other metals, but many salts of the metal find daily use, especially the oxides, the basic carbonate (white lead), the acetate, chromate, and sulphate.

Those who work with lead in the metallic state and in alloys acquire the toxemia in one or two ways. First, by means of getting the toxic agent on the hands and conveying it to the mouth with the food, etc. A second way occurs in those who handle the lead in the molten state. In this condition, fumes are given off and the molten surface quickly becomes coated with a suboxide which is one of the most toxic of lead compounds. Workers who are subject to these dangers are principally pipe-makers, shot-foundry-workers, bullet-makers, and those in lead-casting works, plumbers, tinsmiths, roofers, file-cutters, engravers, braziers, brass-workers, printers, and typesetters. Other poisonings

from metallic lead have been described in persons who have drunk from vessels cleansed with lead shot, and in a few rare instances the poisoning has been traced to the presence of a lead bullet in the body.

Poisonings from the salts of lead occur most commonly among painters from white lead, dye-workers from the soluble salts of lead used as mordants, glass- and enamel-workers, paper-makers where lead salts are used to give weight and "body" to the paper, porcelain-workers, especially kiln-workers, where lead salts are used to furnish the glaze, storage battery and electric accumulator-workers, and in fur-workers where the fur and hair are boiled with lead salts. More unusual forms of poisoning have been observed in seamstresses from putting lead treated thread into the mouth. Flower-makers (Charcot and Yron) and rubber-workers (Putnam). Again the use of lead paste to remove hair and the presence of lead chrome in foods have been known to cause lead poisoning symptoms. Recently twenty cases of lead poisoning occurred in the Brooklyn Navy Yard among men who were engaged in chipping lead paint from the sides of old iron ships.

Clinical Forms.—We are interested here only in the effect of lead on the nervous system, as the general aspect of lead poisoning cannot be considered. A history of lead colic, the presence of a lead line on the gums, and the blood picture of lead poisoning are always important points in the diagnosis, but not always indispensable. The same may be said of the presence of lead in the urine.

Peripheral neuritis is the form of nervous system lead intoxication most commonly seen. This is the usual toxic neuritis, which may be general or partial, with the pains, paresthesias, loss of reflexes and paralyzes that go with peripheral neuritis of any etiology. The most usual and typical neuritis is the local neuritis especially that which affects the upper extremities. If only one arm is affected, it is usually the right. Pain is often the first symptom. This is quickly followed by weakness of the extensor muscles of the hand, beginning generally in the radial region. From this time on the progress is slow, and it goes on eventually to complete paralysis with wrist-drop and muscular atrophies with reaction of degeneration in the paralyzed muscles. Fibrillary twitchings have been observed in the paralyzed muscles in a few cases. The course of the condition is very chronic and without proper treatment stubborn and disabling contractions develop. Unless the toxic agent is removed, other parts of the body become similarly involved and a multiple neuritis develops.

Of the central nervous system poisonings due to lead, we have a large number of illy defined conditions which are grouped under the general heading of encephalopathia saturnina or lead encephalopathy. In an article on treatment, one can hardly hope to differentiate these complex states, so it must suffice to but mention them here. The primary lesion in all cases is, in all probability, a sclerosis of the brain vessels, and the extent and location of the sclerotic process accounts for the nature of the picture. Many of the cases begin with coma and delirium and from this many different conditions develop, among which

are general cerebral arteriosclerosis with apoplexy, prolonged comas, sometimes terminating fatally — “coma saturnina,” epileptiform convulsions, at times going on to status epilepticus, other types of convulsions which may resemble those of hysteria and deliria, with violent hallucinations, resembling at times delirium tremens (mania saturnina). Again one may see hallucinations with unclouded consciousness or a clinical picture resembling arteriosclerotic dementia. Elsching reported some cases with retrobulbar optic neuritis.

A peculiar and little recognized type of lead encephalopathy has been described by Maas. The lesion in these cases has been an internal hydrocephalus with serous meningitis and ependymitis as the primary cause. The symptoms these cases have presented have been in the main those of internal hydrocephalus or of brain tumor. In fact most of these cases had been originally diagnosticated brain tumor. In all probability, many of the cases of so-called “pseudobrain tumor” belong to this type of lead encephalopathy. The most prominent clinical features are severe headache, vomiting and slowly progressive optic neuritis. Some cases have shown the Babinski sign on both sides with increased reflexes, but truly localizing signs are absent.

In the spinal cord changes due to lead poisons have been described but rarely. There is a type of lead pseudo-tabes known, and it is probable that there are cases of progressive muscular atrophy which rest upon a lead foundation.

Prophylaxis.—The general remarks on prophylaxis in the introduction to this chapter apply to the lead intoxications. Removal from the source of trouble is the best measure of all, but unfortunately not always practicable. For years there has been a superstition that the use of dilute sulphuric acid (the so-called “sulphuric acid lemonade”) will prevent lead poisoning, and its use is still practised in many places. The theory upon which this is built is the insolubility of lead sulphate in water. It was hoped by this means to change the ingested lead into the insoluble sulphate and so prevent its absorption. The fallacy of this idea is now known. While lead sulphate is not soluble in water, it is known to be actively soluble in a hydrochloric acid solution of peptones such as exists in the human stomach.

Personal cleanliness and the washing of the hands after work is a matter of great import. However, the habit among painters of washing the hands in turpentine to remove lead bearing paints should be actively discouraged. While the lead salts are certainly removed by this means, the action of the turpentine on the skin is so injurious that it makes the roughened skin more susceptible to the action of the lead and more in a condition to absorb it. All lead-workers should be encouraged to immerse their hands in a solution of potassium sulphide before washing. This converts all the lead salts to the black insoluble sulphide, which must be scrubbed off entirely before the hands look clean. This calls the attention of the worker to the lead on his hands and prevents the overlooking of considerable quantities of colorless lead compounds when the washing is done carelessly.

Treatment.—GENERAL CONSIDERATIONS.—The results of the treatment of acquired lead intoxications are none too gratifying except in the neuritic forms. Having stopped the further ingestion of lead, one must attempt to accelerate its elimination as far as possible. As we have seen, the elimination of the heavy metals takes place principally through the intestines, with the kidneys and skin as secondary exits. The first duty then is to favor intestinal elimination. Every patient at the outset should have full catharsis with calomel up to 10 grains, followed in three to five hours with an active saline cathartic, preferably magnesium sulphate $\frac{1}{2}$ to 1 ounce. Thereafter the patient should receive $\frac{1}{2}$ to 1 ounce of magnesium sulphate every morning until one is satisfied that elimination has been completed.

To liberate the poisons stored in the liver, kidneys, and tissues there is no better medium than the iodides, especially those of sodium and potassium. It is no uncommon observation that the use of large doses of iodide in lead intoxication is frequently followed by a marked exacerbation of the symptoms. This is due to the setting free of large amounts of the poison into the general circulation from the storehouses of the liver and other organs by the iodine salts. In these organs the lead exists as a more or less inert substance, but when it is released into the general circulation it acts on the injured nervous system just the same as more absorbed lead would. Hence, it is always wise to begin with small doses of the iodide salts, *i. e.*, 5 gr. *t. i. d.*, and increase, gradually until the patient is taking 60 to 75 grains (4 to 5 grams) *q. d.*

With the lead liberated into the circulation by the use of iodides, one may accelerate its elimination by means of the kidneys and possibly the skin. Of the diuretics, the salines are of most value. The familiar A B C diuretic of the New York Hospitals, which consists of $7\frac{1}{2}$ grains each of the acetate, bicarbonate, and citrate of potassium to the $\frac{1}{2}$ ounce of water, given every three or four hours, is an excellent preparation. When used in conjunction with large amounts of water, the diuresis is plentiful and satisfactory. The use of irritant diuretics such as those of the uric acid series is to be avoided.

Of diaphoresis much less can be said. As an eliminant the skin is rather a poor thing, but nevertheless it should be used as an adjuvant to the kidneys. The use of ingested diuretics, such as pilocarpine, is ordinarily not advisable. Hot baths, steam baths, and hot packs are the best diuretics we have. Von Jaksch recommends the use of warm baths, such as those of Baden and Aachen. The author has found the following routine of value together with the procedures outlined above. A hot tub bath 100° to 110° F. every morning for one hour and a wet hot pack at 120° F. every evening for forty-five minutes. Some authors favor the use of electric light baths, but it is difficult to see in what way they surpass the hot pack. Again, hot sulphur baths are frequently recommended, and here again one can see very little to recommend them except the heat. The skin at best eliminates very little of the toxic agent, and the external application of dilute sulphur salts can hardly aid to any extent.

TREATMENT OF LEAD NEURITIS.—It is hardly necessary to enter here upon the treatment of lead neuritis. The treatment of the neuritis *per se* does not differ from that of the treatment of neuritis from any other cause, and this subject has already been expounded in another chapter.

TREATMENT OF LEAD ENCEPHALOPATHY.—The results in the treatment of lead encephalopathy has so far been none too happy. When one is dealing with a condition of more or less extensive brain vessel change, there is little to encourage a hopeful outlook. Segelkern, however, obtained good results in a case of convulsions with following coma for thirty-six hours in a lead-worker by the use of the lumbar puncture. In this case he found the fluid to be under high tension and after the removal of 60 c.c., consciousness returned and the patient showed no further symptoms of his trouble for several weeks. Elsching also recommends puncture and the withdrawal of fluid up to 60 c.c. in the cases of optic neuritis due to lead.

Maas, in his series of peculiar cases of internal hydrocephalus, recommends puncture of the corpus callosum for the complete relief of the symptoms. The technique of this operation is described in the chapter on Surgery.

Besides the special treatment directed toward the elimination of the lead, one must not forget the general tonic measures invaluable in all chronic diseases and in convalescence. The usual routine of fresh air and forced feeding with milk and eggs should never be neglected.

Arsenic.—Incidence.—Nervous system diseases due to arsenic are much less frequent in occurrence than those we have just been considering. In the first place, the toxicity of arsenic is so much greater than that of other poisons that workers are more careful in its use and more laws have been passed to restrict its employment in the trades. Again, the acute poisonings are so severe and not infrequently fatal that fewer workers live long enough in the arsenic trades to acquire chronic poisoning manifestations.

The copper salts of arsenic are the ones of most common usage, and in the past these have been the bases of practically all the green paints. With legislative activity their use has been more and more restricted, until at present one seldom finds arsenic paints in use. The best known of these green pigments are Paris or Schweinfurt green, a double acetate, and arsenite of copper and Scheele's green, the arsenite of copper. Besides their use as paints, these substances have been employed as insecticides especially on potato plants and trees and have even been added to canned green vegetables to preserve the color. The physician very seldom sees arsenic poisoning from this source in the present day, as its use in foodstuffs and other articles of general use is forbidden in all civilized countries.

Arsenious acid finds a very restricted use in industry. Probably taxidermy is the only trade where this substance is used to any extent. Here the arsenites of potassium and sodium combined with soap and gum are often used to stuff animal skins on account of their preservative

action. Arsenic acid has a somewhat wider application and is used in dye works as a mordant, in tanneries for removing hair from hides, and in fireworks factories in the manufacture of white fire.

A survey of the etiology of chronic arsenical poisoning would be incomplete without a reference to the famous epidemic of "beer poisoning" in the North of England, in 1900; the epidemic was one of a painful neuritis with herpes going on to total paralysis. The causative factor was finally traced to arsenic in cheap beer, which was extensively drunk in this section. Analysis of the beer showed arsenic to the amount of 0.14 to 0.3 grain per gallon. The arsenic came in with the sugar used in the fermentation. This was invert sugar, and the inversion had been done by means of sulphuric acid. The sulphuric acid had been made from pyrites mined in Spain. It was found that the original source of the arsenic was here.

Clinical Pictures.—In the nervous system, arsenic intoxication is restricted to the peripheral nerves, although mental signs have been observed. Frequently, arsenical palsy follows an acute intoxication with gastro-intestinal symptoms in from ten days to four weeks. The lower extremities are usually the first affected, giving drop-foot, loss of reflexes, and ataxia. Herein it differs from lead neuritis, which, as a rule, affects the arms and legs. In lighter poisonings there is mono-neuritis with herpes; in severer cases the neuritis may be general, but the cranial nerves are always spared. The reflexes are lost; sensation is impaired, the pains are very severe, and even passive motion causes severe pains. The nerve trunks are exquisitely tender. In very severe cases memory disturbances have been noted, and in some the typical Korsakow polyneuritic psychosis. Some severe cases have been described as "neurotabes," but it is doubtful if the spinal cord is ever involved.

Treatment.—The general principles of prophylaxis and general elimination treatment, which have been outlined in the section on Lead, apply equally as well to arsenic and potassium or sodium iodide, and should be given as in lead poisoning, but it is doubtful if they are of as much value. Raymond advises the use of warm sand and water-baths for the severe pains of arsenical neuritis.

Mercury.—Like arsenic, mercurial poisonings are much rarer in the present day than formerly. The bad effects of mercury upon its workers, while not as severe as those of arsenic, have nevertheless been of sufficient moment to attract the attention of legislatures and occasion restrictive laws. Unlike arsenic, it is difficult to find a substitute for this metal, so that its use in the trades has not perceptibly diminished. The liquid nature of the metal at ordinary temperatures facilitates its ingestion both in its normal state and on account of its easy volatility.

In the industries, mercury is used much more often in its metallic state than in the form of salts. The principal industries in which workers are exposed to mercurialism are the refineries of mercury ores, mirror factories, thermometer, barometer, and mercury suction pump manufacturers; electric battery making, where the metallic mercury is used to

form amalgam with the zinc and also some of the salts are employed; match factories, where mercury salts are used now less extensively than formerly; goldplating-workers; and in the manufacture of felt and silk hats, where salts of mercury are used in mordants and to give weight to the felt.

Clinical Picture.—The sore throat, salivation, and diarrheas of acute mercurial poisoning are too familiar to require mention, but the chronic effects of mercury on the nervous system are much less understood. Of peripheral nervous system affections there is no regular type, and frequently the neuritic symptoms are restricted to the severe pains, often shooting in character, which are general in distribution. In a few severe cases a true amyotrophic multiple neuritis has been described. Mercury also has an undoubted effect upon the central nervous system. The most frequent manifestation is the mercurial tremor, which somewhat resembles that of paralysis agitans in some cases and that of multiple sclerosis in others. In more severe cases, tonic spasms or even general convulsions have been observed. In the mental sphere "mercurial erethism" is known. This is a condition of abnormal irritability, with psychic unrest and dizziness, stuttering, disturbed sleep, timidity, and general muscular weakness. In more profound poisonings this condition may develop into a wild delirium, with more or less transitory hallucinations. Practically nothing is known of the pathology of these central nervous system affections.

Treatment.—After absorption most of the mercury is taken up by and stored in the kidneys, and after these come the liver and spleen as storehouses. In its elimination from the body the intestines do the bulk of the work with the kidneys as very minor adjuvants. Small amounts of excreted mercury have been found in the saliva and still less in the sweat.

In the treatment of mercurialism we see the necessity of encouraging elimination by the intestines. To this end the saline cathartics should be used as has been outlined in the section on Lead Poisoning. To favor the release of the stored metal from the organs, we have no better drugs than the iodides, although here their value is very much less than in the case of lead. In fact, it has been doubted whether their value is at all appreciable. Unlike the case in plumbism, there is no necessity for beginning with small doses in fear of increasing the symptoms. Sodium and potassium iodide may be given up to the point of tolerance, probably hundreds of grains a day, as has been the common procedure in the treatment of syphilis for many years. It is doubtful, however, if there is anything to be gained by such enormous dosage, and in all probability just as much good is to be expected from the use of 45 to 60 grains (3 to 4 grams) every day. Other eliminative measures may be tried—diuresis and diaphoresis by means of hot baths and packs—but too much must not be expected from them. Hot sulphur baths are recommended by some authors, but in them the heat is undoubtedly of much greater value than is the sulphur.

The treatment of mercurial neuritis does not differ from that of

neuritis of any other etiology. The prognosis of the central nervous system affections is not a brilliant one except in the case of the acute delirium. In any event the patient must stop his work and be removed from the source of his intoxication. Exercises, life in the open air, forced feeding, sums up the therapeutic measures open to use. For the mercurial tremor, massage and resistance movements may prove of value.

Copper.—There have been a few cases of neuritis reported in brass-workers which have been attributed to copper. The clinical picture was that of a peripheral neuritis affecting the lower extremities giving weakness and ataxia with loss of knee-jerks, but no loss of pupillary light reflex. The condition has been variously described as "metal turner's paralysis" (Walton and Turner) and "pseudo-tabes cuprica" (Suckling). In one case a green line was seen on the gums like the well-known gray line in lead poisoning. In nature this does not differ from any other form of metal toxic neuritis, and is treated in the same manner.

Manganese.—Embden and the author have described a type of poisoning occurring in workers in manganese. Embden's cases occurred in workers in a manganese dioxide grinding mill and those of the author in workers in a separating mill connected with a zinc mine. The ore from this mine contained considerable amounts of manganese. In both instances the poison was taken in the form of dust.

The clinical picture resembles that of lesions of the cerebellar mechanism of the central nervous system. Gait disturbances, especially asynergia, retro- and propulsion, are the most striking features. The patients are unable to walk down hill without running or falling forward. Reflexes and sensations are undisturbed. The face frequently takes on the mask-like character of paralysis agitans. Coarse intention tremor of the head, body, and limbs is common. In some cases a tendency to impulsive laughter is seen.

The prognosis as far as life is concerned is good, but that for recovery very poor. Some cases have improved on being removed from the dust, but none has recovered: No form of special treatment has been found in the least efficacious.

DANGEROUS TRADES: NON-METALLIC SUBSTANCES

Phosphorus.—The use of phosphorus, one of the greatest sources of industrial disablements, has been of late years very much restricted by legal enactment. Although restricted practically to one trade, that of match-making, yet its toxic effect is so great that it has been accountable for as much suffering as any other substance. Industrial phosphorus exists in two forms: yellow phosphorus, which is soluble and so very volatile that it must be kept under water, and the amorphous red phosphorus, which is practically insoluble and not volatile. Practically all the chronic poisonings come from the use of the yellow variety.

Fortunately the old "parlor match," made of yellow phosphorus, has almost disappeared in the last ten years. They were dangerous not only to the makers but also to the users, for many cases of poisoning and death have been reported in children who have swallowed the heads of matches in play. The number of suicides accomplished by means of phosphorus matches is not inconsiderable. Through legislative activity and popular enlightenment these dangerous articles have been largely superseded by the "Swedish" or safety match in which the non-poisonous red phosphorus is used, which is spread over the side of the box in a paint, and the tipped-head match which contains no phosphorus. Besides its use in the manufacture of matches, phosphorus is used in one other industry, the making of a form of rat poison: the kind whose use is said to "make the rats go out of the house before dying."

Yellow phosphorus is taken into the body mostly in the form of vapor through the lungs, although probably much is swallowed by match-workers. It is only slightly soluble in water, but in the presence of any oil it becomes readily soluble and easily enters the blood, in which it exists in its native state. The excretion of phosphorus is still a matter of doubt, but probably it escapes from the body in the form of an organic compound in the urine.

Clinical Picture.—Phosphorus intoxications affecting the nervous system are so rare that their existence has often been doubted. Henschen has reported seven cases with neuritic symptoms occurring after the absorption of phosphorus coming on in from two days to one month after the poisoning. The main symptoms were severe pains, cutaneous hyperesthesia and hyperalgesia, and great sensitiveness of the nerve trunks.

Treatment.—Henschen, who is the only author reporting phosphorus neuritis, makes no mention of any special form of treatment. As so little is known of the fate of phosphorus in the body and the moment of its elimination, it is impossible to accurately facilitate its passage from the body.

Carbon Bisulphide.—This volatile foul-smelling liquid has found industrial use as a solvent for various substances: phosphorus, iodine, fats, oils, tar, rosin, rubber, and gutta-percha. In the present day its use is practically restricted to the rubber industries, in which many men are employed. Here practically all the cases of poisoning occur among the workers in the vulcanizing rooms, where the bisulphide is volatilized by heat. Absorption takes place through the lungs.

Clinical Picture.—Peripheral neuritis is practically the commonest form of carbon bisulphide disease, and of this three different forms have been described:

1. Mononeuritis.
2. Symmetrical polyneuritis.
3. Pseudotabes.

Carbon bisulphide mononeuritis occurs usually in the arms, either in the median or ulnar nerves. It is a typical neuritis, with pain, and

atrophies of the hand muscles have been described. This form of neuritis has also been observed in the optic nerves (Remak), but never in any other of the cranial nerves. The symmetrical polyneuritis occurs in the lower extremities. It is of the usual type of neuritis, with pains and paresthesiæ, loss of knee-jerks, and drop-foot.

The pseudotabetic form is possibly a form of neuritis, but Stadelmann considers it to be due to the central nervous system involvement. This author described two cases of patients who came from a vulcanizing room, and they showed great muscular weakness, with tremor and fibrillary and convulsive twitchings in the muscles. Besides this there were Argyll-Robertson pupil, diminished reflexes, ataxia, positive Romberg, diminished sensibility, and urinary incontinence.

On the mental side different pictures have been observed. Charcot saw psychic symptoms in 87.5 per cent. of his cases. In some these manifestations were confined to headache, dizziness, dulness, and extreme fatigue. Other cases have difficulty in speaking and some loss of memory. One case had amblyopia. Many of these cases had been diagnosticated hysteria, as have so many other obscure conditions. In severe cases psychoses, very similar to a manic-depressive psychosis, are described. Other authors speak of dementing and paranoid states from carbon bisulphide poisoning.

Prophylaxis.—As in every other form of industrial poisoning, prophylaxis should be the watchword, as the treatment of the acquired poisoning is not encouraging. All vulcanizing rooms should be well ventilated so as to carry off the injurious fumes as rapidly as possible. No man should be allowed to work in a vulcanizing room for more than four hours a day or longer than one week at a time. It should be the routine in all the works to have all the vulcanizers examined by a physician every week. As soon as any suspicious signs develop the workers should be forced to find some other trade.

Treatment.—The prognosis in carbon bisulphide is good for life but poor for recovery. The treatment must be merely symptomatic, as nothing is known of the metabolism of the poison. Excretion probably takes place through the lungs, as in the other gaseous substances. Of the special treatments, one author advises the use of large doses of quinine, but very little clinical data has been collected to attest to its value. Another advises small doses of phosphorus, but this procedure is, in all probability, worse than doubtful.

Cyanides.—Collins and Martland have reported a case of polyneuritis occurring in a silver-polisher employed in a hotel. The method used was to dip each article in a solution of potassium cyanide and then dry it. The man had his hands in this solution most of the day. The clinical picture was that of an extensive multiple neuritis with great muscular atrophy but little sensory changes. In experiments in rabbits the authors found the anterior horn cells of the cord degenerated as well as the peripheral nerves. No special form of treatment was used in this case.

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DANGEROUS DRUGS

Drugs Inducing Acute Psychoses.—Delirium.—There are certain drugs which when taken in sufficiently large quantities over a considerable period of time induce acute psychoses of an active delirious type. These drugs belong among the hypnotic and sedative groups. The bromides are accountable for many of these psychoses, undoubtedly more than is generally recognized. This type of delirium has been

known to have been induced also by antipyretics, such as acetanilide and phenacetin; hypnotics, such as sulphonal and trional; and sedatives, such as belladonna, hyoscyamus and its alkaloids. The author has seen a severe delirium resulting from the use of bromo-seltzer to the extent of four ounces daily. The causative factor here is largely acetanilide.

The metabolism of most of these poisons when taken into the body is very poorly understood, save from the purely theoretical sides, where the studies of Meyer, Overton, and others have shed much light upon the relation of narcotics to lipoids. That of the bromide salts, however, has been accurately worked out practically by Laudenheim. His work throws so much light upon the bromide deliria in special and upon toxic deliria in general and offers so clear a *rationale* for treatment that it may be quoted to advantage in full. He sums up his conclusions in five headings: (1) Bromide salts given to a bromine-free organism are not at first excreted in the same amounts as they are absorbed. For a long time—at least one week—the greater part of the ingested salt is stored up in the body. Only after the formation of “bromide depots” of sizes varying with the individual (up to 52 grams) is an equilibrium between the intake and output established. Under abnormal conditions—polyuria—the output can, in exceptional cases, exceed the intake (proportionately). (2) The stored-up bromide salts remain for the greater part dissolved in the lymph. (3) The primary cause of the symptoms of bromide poisoning must be sought for in an insufficiency of the renal and cardiac action induced by the bromide salts, for, in urinary retention, the bromide is retained together with the other constituents of the urine. (4) In individuals whose body fluids before the use of bromine were very poor in chlorine (anemias, cachexias, etc.) there is a specific tendency to excessive bromide retention and the chlorine is excreted from the body. Here bromism comes on relatively early and from small doses. (5) By the addition of large amounts of sodium chloride the bromine excretion is considerably increased and thereby the “bromide depots” are decreased.

While little is known of the other drugs of this group one might easily assume that their action is not unlike that of the bromides, especially in regard to the storage in the body and their excretion, although it is very doubtful if the close relation to the chlorine content of the body obtains in any other drug than the bromides.

The dosage required to produce a drug delirium naturally varies within wide limits. In the case of the bromides the delirium, according to Meyer, may result from the use of 4 grams (60 grains) of the drug. Again in other cases, as every physician knows, 90 to 100 grains may be given every day for many months without causing any delirious symptoms. The same holds true in the case of the coal-tar antipyretics. The patient previously mentioned with the delirium from bromo-seltzer took an equivalent of 80 grains of acetanilide for over three months before delirious signs developed.

Excretion of these drugs takes place for the greater part through the urine. The intestinal tract probably also excretes a little. The bromides

are eliminated to a very slight extent through the skin, which accounts for the bromide rash so often seen in patients taking large amounts of these salts. The signal absence of a bromide rash in all the reported cases of bromide delirium is added evidence of the fact that faulty excretion is the foundation of the trouble. Belladonna and hyoscyamus are excreted to a small extent in the urine, but much of the active alkaloids of these plants is stored in the body and possibly reduced in the organs.

Clinical Picture of Drug Delirium.—The onset of the condition is usually rather sudden following a period of dulness and apathy. The mental picture is that of the true toxic delirium with confusion, hallucinations of sight and hearing, distractibility, tendency to flight of ideas, memory anomalies, fabrications, misidentifications, and paraphasia. This last is an important diagnostic point. On the neurological side a number of anomalies are met with. The commonest are increase and inequality of reflexes, pupillary changes, and general cutaneous hyperesthesia. At times before the onset the patient's speech changes, and it sounds as though the tongue were too thick for the mouth. In bromide delirium, Seguin has described a "bromide breath"—a peculiar, sweet, fetid odor to the breath.

Treatment.—The principal points in the treatment of the drug deliria are to stop further absorption and increase elimination of the toxic agent. At first all drugs must be stopped, for when more than one drug has been given it is difficult to signal out the culprit. In elimination, as previously shown, the kidneys bear the brunt of the labor. They are our main hope. The saline diuretics are by far the best. The familiar A B C diuretic consisting of $7\frac{1}{2}$ grains each of the acetate, bicarbonate, and citrate of potassium in $\frac{1}{4}$ ounce of water, given every three or four hours, together with large amounts of ingested water, is excellent for increasing renal elimination. The use of the irritant diuretics of the uric acid series is hardly to be recommended in these cases of urinary suppression.

As an adjuvant to the kidneys the skin should be utilized. Diaphoresis is best accomplished by means of heat. A good routine is to give a hot tub bath for one hour every morning and a hot pack at 110° F. for forty-five minutes every night. To favor elimination by means of the intestines the saline cathartics are the most valuable; an ounce of magnesium sulphate every morning keeps this portal of exit well open.

A difficult problem in the handling of these cases comes in the choice of a sedative at the height of the delirious excitement. It is frequently necessary to quiet the noisy and excited patient and to give him the necessary sleep. The best sedative in all excited cases are continuous hot baths and hot packs. The routine hot pack at night not infrequently serves as a sedative and insures sufficient sleep. When these measures are not possible or inefficient one must use drugs. One should not use the bromides, chloral, sulphonal, or hyoscyne, as they not only augment the trouble but are dangerous. Morphine and the

opium derivatives are worse than useless in excited cases. Undoubtedly the best drug to use in this case is paraldehyde. This drug belongs to the alcohol series, and is not only more quieting but is more easily eliminated than the others mentioned, being thrown off in part by the lungs. Doses of 1 to 4 drams three or four times a day are all that is ever necessary.

The diet during the delirium must consist of liquids—milk, broth, and eggs—and the more one can induce the patient to take the better. When the patient refuses to take food, he must be fed with a stomach tube, just as in the case of any resistive delirious or psychotic patient. When the delirium has subsided the usual reconstructive measures must be employed—life in the open air, exercise, and forced feeding.

A special form of treatment in the bromide deliria, which may also be of value in those from other drugs, is the use of common salt—sodium chloride. Laudenheim has shown how the increased absorption of sodium chloride favors the excretion of the bromides, and on this theory it is given. At least 90 grains (6 grams) a day should be given in all cases where there is no tendency to urinary retention. It may be given in milk, water, or in capsule.

The Habit Forming Drugs.—Opium and its Alkaloids.—Cocaine.—

Drug habits belong to human nature in general rather than to any race or type if one includes the drugs in universal daily use—coffee, tea, and alcohol. While these must be included among the drug habits, they are not usually considered such, since their use causes symptoms calling for treatment in only an infinitely small percentage of users. Opium and cocaine and their products form pernicious habits and cravings in their users, ruin their lives, and induce psychoses. These habits are the ones the physician is called upon to treat.

The use of opium is very ancient in the far East, and today the opium habit is the one most frequently seen and treated. Opium may be taken by inhalation, smoking, eaten in the raw form, or as one of the alkaloids or by hypodermic injection in the form of morphine or some other alkaloid. This last is by far the most common practice in the present day. Morphine is the drug most commonly used, but there are many cases of habit contracted from the use of codeine and heroine. The doses taken by old habitués are often enormous. De Quincey took 8000 drops of laudanum a day. Rosenberger reports a case who took as high as 60 grains of morphine every day. This patient would swallow 50 half-grain tablets at once, washing them down with a water solution of chloral and sodium bromide. Jelliffe reported the taking of 90 grains of morphine daily by hypodermic. People taking 15 to 20 grains of morphine a day are often seen.

The cocaine habit is one of more recent date, although the natives of Peru and Bolivia have chewed coca leaves for centuries to prevent fatigue. As a single habit this is not as extensive as is the morphine habit, but in many cases the two exist in the same individual. It is rarely as serious as morphinism, and is the more easily broken habit of the two.

Clinical Picture.—The clinical picture of chronic morphinism is so varying and exists in so many stages that space does not permit of its description here. In the early stages it is almost impossible to detect, as the patient is so successful in hiding it. Later, with the gradual health deterioration and the increase of the grip of the habit, the moral sense is dulled and the patient is more open about his affairs. With the increasing dosage the patient becomes irritable and excited, and still later real psychoses develop. Practically the same thing happens in the case of the cocaine habitué, but here the sedative action is not so great, the pupils are dilated, not contracted, and the loss of weight and emaciation is much more extensive.

The cause for the taking of a drug is practically always said to be pain. When so such pain is often of a severe and lasting character, such as that of gastric ulcer, the neuralgias (principally trigeminal neuralgia), neuritis, sciaticas, the pains in the crises of *tabes dorsalis*, headache, and *puritus ani*. In morphine the patient finds an easy and quick relief from his trouble. It soon becomes necessary, however, to increase the dose to get relief. Later, when the patient tries to stop the drug taking, he is soon overcome with such pains, weakness, and discomfort (abstinence symptoms) that he gladly goes back to his drug for relief. He has now become a habitué or "drug fiend." He realizes his slavery and dependence on the drug. If his strength of purpose is sufficient he may now be able to break off the habit by making an enormous sacrifice and bearing with much pain and discomfort for a short time. More often he goes on with his habit, secretly indulging in it, and jealously guarding his secret from the world, especially from those nearest and dearest to him. Little by little the dose necessary for relief creeps up, and as the toxic effect becomes more marked he increases the dose by jumps in an attempt to bring back his feeling of well-being. It is common to everyone to wish to "feel fit" all the time, especially in this Twentieth Century active living. Every morphinist promises himself that he will give up the habit "some day," but each day that comes is not "the day," and he feels that he must keep up for the morrow. Hence the rapid increase of the drug in an attempt to regain thereby the will and energy which the drug itself has killed.

When he finds that his old friend and enemy is unable to restore him as of yore he turns to anything that may help him out of his difficulty as the proverbial drowning man grasps at the straw. Here is where the patient acquires the cocaine habit in addition to what he already has. This is also the turning point, for now the patient begins on the downward path to the state of the pitiable wreck bereft of will and energy which ends in a psychosis. In this stage the patient is no longer able to conceal his habits and must acknowledge it; his moral, ethical sense has been numbed and he will stoop to any means to obtain his drug. If he finds his usual surroundings unsympathetic he will seek other fields for companionship and associate with other unfortunates like himself. When he reaches this stage, friends and relatives frequently

take charge of him against his feeble will and bring him to a physician for treatment. In all these cases it is found that the patient is taking a good deal more of the drug than is necessary to keep him comfortable. The excess—frequently one-third more than is necessary—represents the unfortunate's attempt to regain his old-time energy and feeling of well-being.

Treatment.—The main thing in the treatment of drug habits is the withdrawal of the drug. This forms the first obstacle. While the patient desires to be freed from his slavery he still more dreads the immediate effects of the discontinuance of the drug. He finds himself somewhat in the position of the man with an aching tooth who wants to be relieved of his pain, but holds back for fear of the pain of the extraction. Although the drug habitué will place himself under the care of the physician or voluntarily enter an institution for cure, he nearly always makes sure of having a large amount of the drug with him before he does so, in order to meet any possible emergency in his old way. Such patients use great ingenuity in secreting the drug about themselves, carrying it in fountain pens, lockets, cigarette cases, pipes, toilet articles, in the lining of their clothes, and even in the body cavities. Before starting a cure at home or in a sanitarium the patient must be divested of all his clothes, given a bath, and moved to a room he has not previously occupied. Fresh clothes, carefully examined, should be given him and every little article of personal use, such as those mentioned above, must be carefully inspected before being given to the patient, to make sure that no drug is secreted in it. One must also make sure that the patient has no money within reach with which to buy drugs or to bribe attendants.

A trained attendant is an absolute necessity in all cases and one must be with them every minute of the time, especially at first. It is worse than useless to entrust this duty to a member of the family, a relative, or a kind friend. Their hearts are too easily touched by the intense suffering of the habitué when his drug is taken from him, and, unable to withstand his cries and pleadings, they frequently give him "just a little" of the drug to ease his obvious distress. When the patient from whom the drug is being withdrawn rapidly says he feels comfortable the physician should be very suspicious. One may be sure in this case that the drug is not being withdrawn in spite of all assurances to the contrary from whatever sources. In choosing an attendant for a drug case one must be sure to get one whose loyalty and sense of duty are above question. There is no doubt that women attendants are much more reliable than men. Their honesty and devotion to duty are apt to be greater.

When it comes to withdrawing the drug there are three generally used methods: (1) Gradual withdrawal. (2) Immediate withdrawal. (3) Rapid withdrawal.

Each of these methods has strong advocates and equally violent opponents. When one reviews the literature on the subject, one is immediately struck with the spirit of contention which pervades the

whole situation. Each author stoutly defends his own methods and more or less violently assails all others. All have in them much to recommend them. Each is applicable to a certain class of cases.

1. **GRADUAL WITHDRAWAL.**—The first thing is to determine how much of the patient's daily drug intake is superfluous. Limit him to the smallest amount which will keep him comfortable. When both cocaine and morphine are used the cocaine is nearly always a superfluous drug and can be discontinued at once. The amount of morphine can frequently be decreased by a third in two days without causing any discomfort. Patients taking ten grains a day can easily be reduced to six in most cases, but from this point on there is difficulty. Any further reduction causes discomfort and collapse if continued.

Brouardel recommends very gradual reduction, lasting over a period of six weeks to two months. He advises keeping the patient in bed during the treatment, as this allows a greater control of him and also prevents accident.

Jennings advises reduction by a quarter, a third, or a half of the original dose over a period of four to ten days. Such rapid withdrawal causes heart weakness, collapse, and stomach symptoms. The cardiac collapse he treats with the use of sparteine in doses of one-third to one-half grain every three or four hours as indicated. This author highly recommends the use of large doses of sodium bicarbonate for the stomach pains and symptoms, which he ascribes to hyperacidity. This he gives by preference in the form of vichy. Purgatives, preferably salines, are a very essential part of the treatment.

Pressy takes months to completely withdraw the morphine. He first finds the minimum necessary dose and puts the patient on this for two or three days. If he is feeling well at the end of this time he makes a small reduction and keeps the patient at this dose until he again feels well. He never reduces the dose to such an extent as to cause anything more than slight discomfort and certainly never enough to require any stimulant. Of course, with this method, it is necessary to keep the patients in a sanitarium and under constant observation. Tonic treatment and nutritious diet are necessary adjuncts to the treatment. When the drug has been gradually reduced so that the patient is taking only $\frac{1}{10}$ grain at a dose it may be safely withdrawn altogether. After this the patient should be kept under careful observation and not allowed to return to his home for some weeks. Frequently, patients will want to go home one week after the last dose, as they feel so well, but this is to be discouraged. Many begin to feel badly again after ten to fifteen days, for a few days, and then become much discouraged, feel that the cure was a fraud, and go back to the drug as their only relief.

An easy way for arranging for the decreasing doses, where the daily amounts taken are not excessive, is by the use of Magendie's solution of 16 grains of morphine sulphate to one ounce of sterile water. This solution can be given by hypodermic in doses of $7\frac{1}{2}$ minims ($\frac{1}{4}$ grain of morphine) as often as is necessary. Every time $7\frac{1}{2}$ minims of the

solution is withdrawn the same amount of sterile water should be added to the bottle. By this means a very gradual reduction can be made in a week or two, and when the solution becomes very dilute, sterile water may be substituted without causing inconvenience.

2. IMMEDIATE WITHDRAWAL.—This method was introduced by Levinstein in Germany and seems to have found more favor abroad than in America. In using this form of treatment the patient must be completely isolated and under very strict observation and care. This procedure is naturally followed by a period of intense suffering and often danger for the patient, and he requires very active treatment. The morphinist whose drug is suddenly stopped becomes frenzied, is hardly responsible for his acts, and will commit suicide or even murder if the opportunity is open to him.

The physician must in every case substitute something for the drug. For this purpose the use of other opium products, cocaine or cannabis indica, is not to be considered for an instant. The drugs usually substituted fall readily into two classes: (1) sedatives; (2) cardiovascular stimulants.

Sedatives.—These drugs are given with the idea of “knocking the patient out” until the period of his reaction is past. Sodium and other bromides are not of much value and the same may be said of trional, sulphonal, etc. Hirt recommends the use of chloral in large doses. His procedure is as follows: After withdrawing the morphine a long sleep, twenty-four hours if possible, is advisable. To insure this he gives 45 to 50 grains (3 to 3½ grams) of chloral on the first and second days, and on the third and fourth days, 30 to 45 grains (2 to 3 grams) of trional, or more if necessary, to allow the patient to sleep most of the time. After the fourth day the crisis is generally over and reconstructive treatment may be begun. The author recommends warm baths followed by cold showers, two to four times a day, beginning on the fourth day. On the fifth day he begins to employ hypnotic suggestion, in sessions of one hour each, three times a day. The use of the lighter hypnotic states is usually sufficient. Hirt considers institution treatment for all drug cases to be necessary and advises its continuance over a period of three weeks to nine months.

The drug most frequently used now as a hypnotic in cases of rapid morphine withdrawal is hyoscine. This method was first introduced by Lott in Texas. The procedure is as follows: Withdraw all morphine at once and start with hypodermics of hyoscine hydrobromate $\frac{1}{100}$ to $\frac{1}{100}$ grain every thirty minutes to one hour. Push the hyoscine to the point of tolerance. If the heart action becomes weak, give strychnine $\frac{1}{10}$ to $\frac{1}{10}$ grain every three hours per hypodermic. At first the patient becomes excited, but later is simply restless, with low mutterings. On the second day give the above dose of hyoscine every one to two hours, enough to keep the patient well under the influence. Use strychnine to overcome cardiac weakness. This dosage is kept up for one week to ten days and then the amount is diminished slightly, giving it every three or four hours until the twelfth or fifteenth day,

when it may be discontinued. The same period of observation, rest, exercise, and reconstructive treatment should follow as in all other methods.

The rationale of the cardiovascular stimulant drugs is different from that of the use of the sedative drugs, for with the stimulant an attempt is made to overcome the harrowing effects of morphine withdrawal by stimulation of the vasotonic system rather than by dulling the consciousness, as in the case of the hypnotics.

Erlenmeyer and Hofman are enthusiastic over the use of camphor as a stimulant, as they claim that its effect on the cardiovascular system is exactly opposite to that of morphine; that is, it contracts the vessels and lessens the size of the heart. The camphor is given by mouth in the dosage of $7\frac{1}{2}$ grains (0.5 gram) one to five times a day. The camphor is used as is necessary to keep the patient fairly comfortable.

Ergot has also been advised in this respect by Livingston. This author considers the agitation and other mental signs following the withdrawal of morphine to be due to vasomotor paralysis. This vasomotor paralysis is seen in the cutaneous hyperemia after the drug has been discontinued. Livingston considers the stomach and mental symptoms to be due to the same vasodilatation. Ergot is, in his mind, the best vasoconstrictor for this use. He proceeds as follows: as soon as the patient is brought under control, discontinue the morphine at once and give him a mercurial purge; institute fluid diet with abundant fluids, and all during the treatment give enough cathartics to cause two or three bowel movements a day. He starts with his ergot treatment as soon as possible; the mixture he employs consists of solid extract of ergot (Squibbs) 1 dram and sterile distilled water 1 ounce; filter, add two drops of chloroform, and shake. Of this mixture he uses 30 minims per hypodermic, two or three times a day in mild cases and every two hours in severe ones. This is continued until the patient feels well.

3. RAPID WITHDRAWAL.—The term "rapid withdrawal" is used to indicate a method intermediate between the gradual and the immediate withdrawal methods. Ordinarily the withdrawal is completed within two or three days. Any number of modifications of the methods here outlined have been used, and in a way come under this heading. It would be hard to outline the number of modifications and combinations of the previously mentioned methods possible.

In recent years a form of treatment which comes under this heading has become very popular and much good is claimed for it. This is the so-called "Town's treatment." This form of treatment was devised and elaborated by Charles B. Towns and Dr. Alexander Lambert, and consists, in the main, of active catharsis and the use of a special mixture. This consists of two parts of the 15 per cent. tincture of belladonna. One part each of the fluidextracts of hyoscyamus and xanthoxylum. When the ten to twelve per cent. belladonna tincture is used the results are never as good. The mixture should be pushed to the point of tolerance in hourly doses. Some people need only

2 to 4 drops while other tolerate 18 to 20. The bottle containing the mixture should be well corked and shaken before using.

Dr. Lambert describes the method as follows:

"A patient addicted to morphine is given 5 compound cathartic pills and 5 grains of blue mass, and six hours later, if these have not acted, they are followed by a saline. After three or four abundant movements of the bowels from these cathartics the patient is given, in three divided doses, at half-hour intervals, two-thirds of the total daily twenty-four-hour dose of morphine or opium to which he has been accustomed. Observe carefully after the second dose has been given, as the amount then equals four-ninths or nearly one-half of the total twenty-four-hour dose. Some few patients cannot comfortably take more than this amount. At the same time with the morphine, six drops of the belladonna mixture are given in capsules. This belladonna mixture in doses of six drops from an ordinary medicine dropper is given every hour for six hours. At the end of six hours the dosage is increased two drops. The belladonna mixture is continued every hour of the day and every hour of the night, continuously throughout the treatment, increasing two drops every six hours until 16 drops are taken, when it is continued at this dosage. It is diminished or discontinued any time, if the patient shows belladonna symptoms, such as dilated pupil, dry throat, or redness of the skin, or the peculiar and insistent voice and the insistence of one or two ideas. It is begun again at reduced dosage after the symptoms have subsided.

"At the tenth hour after the initial dose of morphine is given the patient is again given 5 compound cathartic pills and 5 grains of blue mass. These should act in six to eight hours after they have been taken. If they do not act at this time some vigorous saline is given, and when they have acted thoroughly the second dose of morphine is given, which is usually about the eighteenth hour. This should be one-half of the original dose—that is, one-third of the original twenty-four-hour daily dose. The belladonna mixture is still continued, and ten hours after the second dose of morphine has been given, that is about the twenty-eighth hour, 5 compound cathartic pills are again given and 5 grains of blue mass. This again, if necessary, is followed by a saline seven or eight hours later. At times when the 'C.C.' pills are not acting well or too slowly, 5 or 6 'B.B.' pills are given two or three hours after the 'C.C.' pills. These 'B.B.' pills are the *pilulæ catharticæ vegetabiles* of the *Pharmacopœia*, with $\frac{1}{10}$ grain of oleoresin of capsicum, $\frac{1}{2}$ grain of ginger, and $\frac{1}{8}$ M of croton oil added to each pill. After these have thoroughly acted at about the thirty-sixth hour a third dose of morphine is given, which is $\frac{1}{3}$ of the original dose. This is usually the last dose of morphine that is necessary. Again, ten hours after this third dose of morphine, that is the forty-sixth hour, 5 'C.C.' pills and 5 grains of blue mass are again given, followed seven or eight hours afterward by a saline. One expects at this time to see a bilious green stool appear. When this appears after the bowels have moved thoroughly, ten or twelve hours after the third dose of morphine, about

the fifty-sixth hour, 2 ounces of castor oil are given to clear out the intestinal tract thoroughly. During this last period, when the bowels are moving from the 'C.C.' pills and before the oil is given, the patients have their most uncomfortable time. Their nervousness and discomfort can be controlled usually by codeine, which can be given hypodermically in $\frac{1}{4}$ -grain doses and repeated if necessary, or some form of the valerianates may help them.

"About the thirtieth hour these patients should be stimulated with strychnine or digitalis, or both. After they are off their drug I have found the tonics which do them the most good are those which contain some form of phosphorus and arsenic. I must reiterate as before the danger of these patients overeating, thus bringing back their withdrawal symptoms due to disturbance of digestion. They have been in the habit of referring all uncomfortable feelings to those of the withdrawal symptoms of morphine, and digestive disturbances feign these withdrawal symptoms. Sometimes, about the thirty-sixth hour, the stools become clay-colored. Some form of prepared ox-gall is most efficient to stimulate further biliary secretion, given in small doses, every hour for five or six doses."

Cocaine.—The treatment of the cocaine habit is a much simpler thing than that of the morphine habit. The distressing symptoms which follow the withdrawal of morphine seldom, if ever, are seen in the cocaine fiend. The method of gradual withdrawal may be used or even that of rapid withdrawal. It is seldom that stimulation is necessary or even sedatives, while the drug is being withdrawn. The Lambert treatment may be also applied to cocainism. Here the procedure is somewhat difficult.

The belladonna mixture and 5 compound pills and 5 grains of blue mass are given simultaneously at the first dose. The belladonna mixture is continued every hour of the day and night the same as with morphine patients. Twelve hours after the initial dose the patients are again given 3 to 5 compound cathartic pills, and at the twenty-fourth hour after the initial dose they are again given the cathartic, followed by salines if necessary, and again at the thirty-sixth hour. After these last cathartics the bilious stool will appear, and by the forty-fourth or forty-fifth hour the castor oil is given. It is never necessary to give cocaine at any time, but strychnine or some such stimulant must be given from the beginning of the treatment.

CHOICE OF THE METHOD.—This survey, although very sketchy, probably will result in confusing the physician and leaving him in doubt as to what method he should pursue. All are valuable in a way, and all have bad points. What succeeds in one patient or with one physician may lead to total failure in another. One may say that on general principles the method of slow withdrawal is easier on the patient, but probably not so likely to produce permanent results as the others. The immediate method of withdrawal is certainly a heartrending procedure. The unfortunate morphinist lives through a perfect hell upon earth until his reaction symptoms are passed. Many authors

describe this treatment as brutal, and probably it is, but it surely makes a strong impression upon the patient! The Lambert treatment is probably the one most used in the present day, and although very active it nevertheless does not have the severe effects of the ordinary immediate withdrawal methods. When morphine is to be withdrawn immediately the use of cardiac stimulants rather than the hypnotics probably assists more toward the patient's ultimate comfort.

The permanency of these cures for drug habits have ever been a source of strife. There is no doubt that the craving for morphine and cocaine can be removed from a patient, but there is also no means of keeping the patient from later going back to his habit. It is an easy way of stimulating himself to bear over an emergency, and with a weak will and a personality which is not used to looking far into the future one may easily see how the patient may drop back into his old ways. I believe this is more apt to occur when the treatment has been done by means of the slow withdrawal method. Here the suffering is not great and the patient does not look back upon his cure with any amount of fear. When the more rapid measures have been used the whole thing represents a very unpleasant experience in the patient's life, and one which he is not anxious to have occur again.

There are cases which are helped in refraining from returning to morphine by a fear that the treatment has engendered in them. There are probably also others who never again undergo a treatment when they have backslid on account of this same fear, and a second taking up of the habit is always the last. They never again wish to be cured.

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CHAPTER XVII

SURGERY OF THE BRAIN AND SPINAL CORD

By ALBERT E. HALSTEAD, M.D.

AND

ROGER T. VAUGHAN, M.D.

History.—The surgical treatment of lesions of the skull and cranial contents dates back to the earliest age of man. Although we commonly think of cranial surgery as the particular acquisition of the last two or three decades its beginnings reach as far back into antiquity as we have any human records. Manouvrier has studied a number of neolithic skulls showing trepanation openings. These were unearthed in the department of Siene-et-Oise in France. Most of the skulls thus trephined show only a single opening, but some show two or even three. The bone is cut through obliquely, the opening in the inner table being much smaller than that in the outer. In some cases multiple puncture openings were made and these then connected. What the technique was, in general, is not difficult to surmise from the appearance of the openings and from our knowledge that no metal tools existed at that time. Flint, stone, wood and bone were the only working materials of our stone age ancestors. Even in those primitive days the various cranial operators lacked uniform technique. In the "temporal decompression" shown in (Fig. 68) the bone was filed away by a rough, file-like piece of flint until the dura (or perhaps the cerebrum also) lay bare. Other operators preferred to saw around in a circle until they reached the dura and then pry up the bone flap with a sharp flint elevator. Others made multiple punctures with a sharp flint gouge and hammer and ended by breaking the bone between these punctures. Further, details of the operations the study of the skulls does not reveal; but that some of the subjects survived the operation is evident from the fact that callus and new bone was formed at the operation sites. In some of the cases the operation must have been performed in tender years since the disturbance in growth consequent to the operation led, in some skulls to marked deformity.

That a certain calloused equipoise and a muscular assistant were the requisites for the surgeon of that day, and some fortitude on the part of the patient, may be surmised. It is possible, however, that some of the operations were performed while the patient was unconscious.

What the indications for the procedure were is easier to surmise than to prove. Rene Lefort states that certain of the neolithic cases of trepanation still show the traces of organic lesions—hydrocephalus, bone necrosis and traumatic lesions.

Paul Broca suggested that the great frequency of trepanation in the neolithic age was due perhaps to a conception that certain intracranial affections, idiocy, insanity, epilepsy, and convulsions were divine attributes and the letting out of the imprisoned spirit or devil a sacred operation which made its subjects an object of veneration; hence his occasional long survival in that rough age. The fact that trepanation, cauterization, and other mutilating cranial operations have been done in historic times among primitive peoples for similar indications lends weight to Broca's view.

The sincipital "T" is another peculiar cranial procedure dating back to neolithic times. (See Fig. 70.)

It consisted of burning a rough "T" into the vertex of the cranium, probably directly through the scalp, by using a hot coal or heated flint. Manouvrier interprets this procedure as a surgical operation and suggests that since it is found chiefly in female skulls it may have been done for hysteria, and under these circumstances it may have been sometimes effective.

Cranial surgery fell into disrepute on the continent of Europe in the times immediately after the neolithic, but other primitive peoples have practised it again in more recent times, as has been frequently recorded. Herodotus states that the Lybian nomads cauterized the scalp for many ailments, and even as a prophylactic measure against disease in general. It has been recorded by Prescott that the ancient Incas of Peru practised trepanation and closed cranial defects by inserting silver plates. In the Muniz collection of Peruvian skulls there are several with trepanation openings, one of which was closed by a silver plate. McGee has written up these skulls in the *Bulletin of the Johns Hopkins Hospital* for 1895.

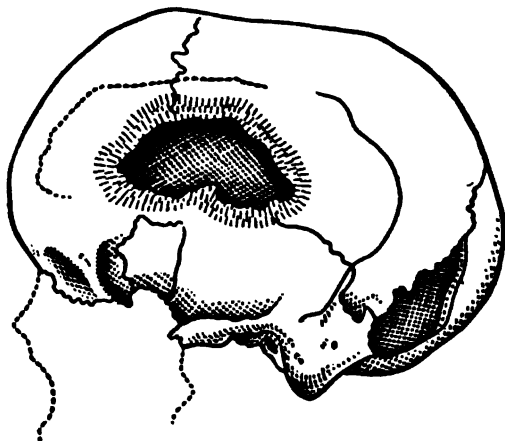
In the time of Avicenna the Arabs in Persia practised cauterization of the scalp for cure and prevention of infantile convulsions. A crude cranial surgery was practised in ancient times in both China and Japan.

Hippocrates did trephining and described two trephines, one circular and auger-shaped, not unlike our present-day instrument. He advised against opening the skull in the vicinity of the sutures (in order to avoid the sinuses) and also gave directions for avoiding the middle meningeal artery and its branches. He furthermore recommended punctures of the ventricles in hydrocephalus, and for this procedure Heliodorus developed special indications. Hippocrates recommended the prolongation and deepening of scalp wounds for exploring the condition of the skull. Galen appears to have paid no attention to cranial surgery and makes no mention of the trephine or its uses. It appears not to have been much practised again until the fifteenth or sixteenth century. A. Paré and Andrea a Cruce (Venice) described a complete trepanation instrumentarium and used an auger-like tool which was armed with a collar to prevent penetration into the brain substance. Mogatus, also in the sixteenth century, advocated the use of perforated gold or lead plates for the closing of cranial defects. Ambroise Paré was strongly opposed to this procedure on the ground that the

human organism would not permit the continued presence of a foreign body in it, a conception upheld also by Galen.

Job a Meckren performed a plastic on a skull defect by implanting a piece of fresh bone from a dog. The implant healed in place. The patient, overjoyed, spread the news of his cure so extensively that it came to the attention of the church authorities, and they took so radical a stand against this unholy performance that the unfortunate patient was compelled to have the offending canine implant removed in order to remain within the pale of the church. The second operation is recorded to have been by no means an easy procedure, so solidly was the bone united.

FIG. 68



(From Auvray, *Malad. du Crâne.*)

Prehistoric skull with trepanation openings, found in France at Menouville, Seine-et-Oise. The formation of new bone about the openings shows that the subject survived the operation. (Manouvrier.)

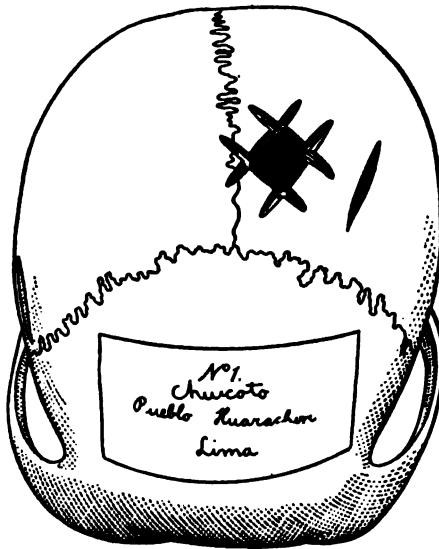
Trepanation for fracture was much practised in the Sixteenth and Seventeenth Centuries and many cases of multiple trepanations are recorded in this connection. Count Philip of Nassau (1664) submitted to 27 successive trepanations at the last of which the point of fracture was discovered! La Ponche (1675) trephined one patient 52 times.

In the following century trephining attained a great vogue in England, owing to the teaching of Pott. In Cornwall, especially among the miners, it was one of the most common of operations on account of the frequent cranial traumatisms.

De Marchettis and Marc Aurele Severin (about 1650) recommended trephining in the treatment of traumatic epilepsy, hydrocephalus, melancholia and migraine. De la Motte (1730) recommended the opening up of apoplectic foci. Corvinus (1747) and Thiebault (1792) the incision and excision of encephalocele. Quesnay (1743) advocated exploratory incision of the brain. Desault, early in the nineteenth century, appears to have been the first to remove a malignant tumor from the brain.

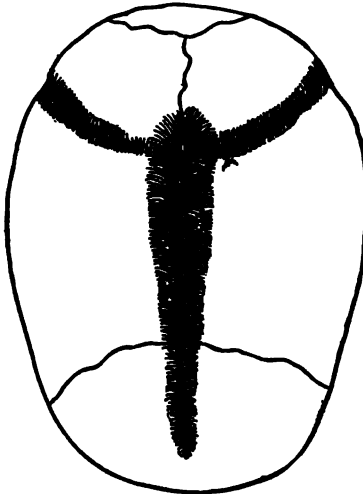
In the early part of the Nineteenth Century trepanation declined in favor owing to the frequent accidents, septic and others, which accompanied it; Dupuytren, Malgaigne, and Nélaton were all opposed

FIG. 69



Trephined skull from Peru (from Munis collection). (From Cushing in Keen's Surgery.)

FIG. 70



(From Auvray.)
Sincipital "T" on a neolithic skull. (Manouvrier.¹)

¹ See Bulletins de la Société d'anthropologie for Manouvrier's article quoted by Auvray, p. 5.

to opening the skull. Leon Lefort states that in the years 1857 to 1867 there were only four trepanations done in all France. The same was likewise the case in England and Germany. It was only after the beginning of the antiseptic period that cranial procedures came to be seriously considered again. In the last thirty years the advances in this field have been far greater than in the thirty centuries preceding. References to the great services rendered in this field by Bergmann, Macewen, Horsley, Starr, Keen, Kocher, Chipault, Doyen, Cushing, Frazier, Krause, Ballance, Hartley and others, will be frequently made in this chapter.

CRANIECTOMY FOR EXPLORATION AND DECOMPRESSION

Indications.—Since almost every operative procedure on the cranial vault has exploration more or less directly as its aim and since, where exploration fails to detect the disease, decompression relieves the symptoms, we find this particular operation the most frequently performed and important in our work. With it may be combined brain puncture; removal of tumor; drainage of abscess; removal of blood-clots; the control of hemorrhage and drainage of the ventricles. Therefore, together with its modifications, it includes nearly the entire field of cerebral surgery.

The conditions for which it is commonly done are the following: (1) tumor cerebri, localizable and non-localizable, and particularly when associated with choked disk and pressure symptoms; (2) brain abscess; (3) intracranial hemorrhages; (4) craniostenosis (particularly tower skull); (5) and rarely hydrocephalus, unless associated with tumor (usually brain puncture is done here in preference to craniectomy); (6) brain syphilis, which does not yield readily to specific treatment; (7) encephalitis in some cases when associated with increased tension and causing optic neuritis or choked disk; (8) edema of the brain in uremia has been considered an indication by Cushing, but is a doubtful one; and (9) in general any condition associated with increased intracranial pressure and swelling of the optic disk demands for the relief of pressure symptoms and optic nerve changes an exploratory operation which may be later turned into a remedial operation, the character of which will depend on whatever pathological condition may be found, or into a decompressive or palliative operation, if anything accessible be found.

Preparation.—In our clinic, on the day before the operation, the patient's head (whole head, if a man; something less than half the head if a woman) is shaved, scrubbed with tincture of green soap on gauze, rinsed with sterile water, then with alcohol, and then with 1-1000 bichloride. A dry dressing is then applied. The scrubbing is again repeated on the operating table just before the operation.

A double layer of gauze (either dry or wet and wrung out of a solution of 1 to 1000 bichloride) is then applied to the entire cranium and

over this the rubber constrictor. We use an India rubber band, one inch wide and eight inches in circumference, which can be obtained of any house handling rubber goods. We make two or three turns with it around the head, which we find is constriction enough. There is no buckle to fasten; it never slips; so that tapes, as used by Cushing, are unnecessary, and the cost is a few cents only. The patient is then ready for the initial incision.

FIG. 71



Author's method of controlling hemorrhage from the vessels of the scalp by means of a large rubber band.

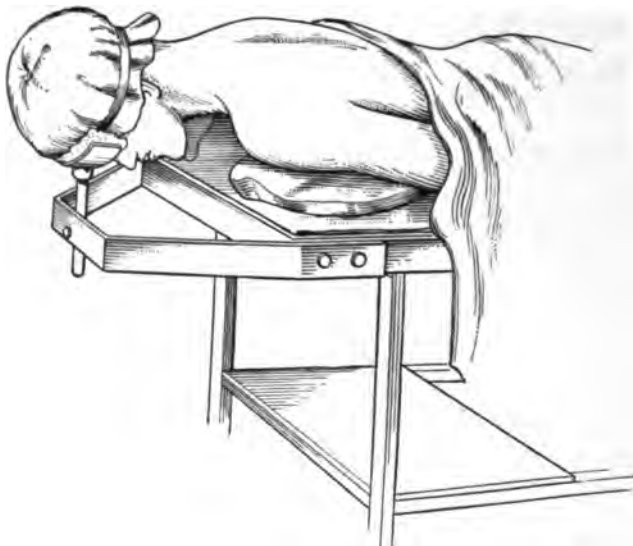
Anesthesia.—Before the patient is brought to the operating room he is given a hypodermic of morphine gr. $\frac{1}{4}$, atropine gr. $\frac{1}{100}$, or if the patient be small, or a woman, $\frac{1}{8}$ and $\frac{1}{100}$ gr. respectively.

Ether by the drop method is, as a rule, the anesthetic of the entire operation. But in some cases, especially when for one reason or another speed is necessary, the anesthetic is started with nitrous oxide and oxygen.

Position of the Patient.—For temporal and motor area operations and in general for all operations on the anterior half of the cranium, the patient lies on his back, with head and shoulders raised, and anesthesia is administered as for other operations about the head and neck. The operator and his assistants are, however, separated from the anesthetist by a sheet, which is held up between the field of the operation and the field of the anesthetist by an outrigger attached to the table.

For suboccipital operations, and frequently for other operations on the posterior half of the cranium, the anesthetic is given and the operation performed with the patient lying on his face, the forehead supported on a head-rest in the form of a bracket attached to the head end of the table, with sandbags under each shoulder. These elevate the chest from the table, permitting relatively unrestrained breathing.

FIG. 72



Curtis-Halstead table (St. Luke's Hospital, Chicago). Sandbags under the shoulders; outrigger for the forehead. Gause over the head secured by a Halstead rubber band.

The anesthetist sits on a low stool and is covered by the laparotomy sheet drawn over the patient's head. The anesthetist, for his own comfort, wears a rubber cap and rubber apron, closely tied about his neck, to protect his clothing.

We do not believe in further incommoding our anesthetist with a phonendoscope, as recommended by H. Cushing, unless it be impossible to secure another assistant, especially for the purpose of watching pulse and respiration. The extra assistant is easily obtained in any large hospital, and the anesthetist has his hands fully occupied and is in discomfort enough without being put to this extra inconvenience.

Occasionally the Matas apparatus is used. Air saturated with chloroform vapor is led into the patient's retronasal space by means of a rubber catheter introduced through the nose. When this apparatus is used the patient is put to sleep by the usual mask method, and just before the operator is ready to begin the nasal catheter is inserted and anesthesia continued with the apparatus. The anesthetist is thus completely removed from the field of operation. As only a relatively small amount of anesthetic can be administered by this method it is

usually desirable to use chloroform instead of ether, especially with large and fat patients and in alcoholics.

The Incision.—The incision is usually marked out on the scalp with iodine just before the double layer of gauze and the tourniquet are applied, so that the still wet iodine stains through the gauze. In locating the incision measurements are usually dispensed with. The eye of the surgeon who is constantly employed in cranial work becomes as accurate as any of the craniometers on the market, and it is moreover always easy to enlarge the opening in any direction subsequently desired.

The incision is semicircular, to form a flap with the base in the direction of the blood supply. The first cut is made down to the lamina externa.

At this point there is always some little hemorrhage from the vessels of the galea. This is usually easily controlled by (1) promptly turning the flap back and exerting pressure on it, providing that an osteoplastic operation is not to be done; (2) by catching and twisting with sharp Kocher forceps, or ligating the "spurters" with fine catgut, No. 1; (3) by pressure by an assistant with gauze along the margins of the incision. If the rubber tourniquet is used, the hemorrhage is insignificant.

The other measures sometimes recommended for stopping this hemorrhage we have never found necessary to use, and believe that in this we are sustained by the best American practice.

The running stitch of Jacob Frank, of Chicago, later modified by Heidenhain and known abroad by the latter's name, would be useful, but is scarcely necessary even though the tourniquet be not used, as, for example, in suboccipital and subtemporal decompressions. To keep these stitches in place for eight to ten days, as Heidenhain recommends, seems to us not only unnecessary, but even dangerous to the health of the flaps, and certainly predisposes to slow healing of the wound and, therefore, to frequent loss of cerebrospinal fluid.

Kredel's plates we have even less use for than Frank's ingenious stitch.

Attacking the Bone.—If an osteoplastic resection is to be made, we proceed at once to bore through the skull. If merely a decompression is desired we first strip back the periosteum with the skin flap.

Bone Instruments.—We believe we have tried them all and have come back to the trephine or De Vilbiss burr, supplemented by the De Vilbiss forceps.

All electric-driven machinery we have discarded—burrs, drills, rotary saws, etc. They are all wrong in principle because they open the skull from without inward and therefore expose the brain to injury from excessive penetration and bone splinters. They are all liable to slip at times, even in the hands of the most experienced men, as our dental colleagues can testify, and cut undesired gashes in the bone and scalp. Moreover, their greater speed is largely offset by their liability to get out of order, thus causing long delays, and leading to

faulty asepsis. Again, it is impossible to sterilize them *in toto*; and therefore slipping towels in the course of long operations are apt to leave non-sterile parts exposed to the surgeon's touch.

The mallet and chisel we have wisely discarded because (1) of the time consumed in entering the skull, and (2) for the reason that we believe the repetition of blows upon the skull may possibly, as claimed by many, produce cerebral concussion.

We have been very well pleased with the De Vilbiss tool. Its chief value is not only in the rapidity with which it can be operated, but in its mechanical construction, which is so adjusted that when the teeth of the trephine strike non-resisting dura they cease to cut, thus making injury to the dura and underlying brain impossible, even when considerable manual force is applied.

The trephine opening is made large enough to admit a De Vilbiss cutting forceps, and with the latter the bone incision is enlarged in the direction of and parallel to the skin incision, or as desired, but keeping well inside the latter, in order to avoid overlapping of the bone and galea incisions.

If an osteoplastic flap is to be made, the bone incisions are continued around to the narrow base of the flap, which is then broken across by prying up the flap with a bone elevator and perhaps also making cuts at the sides with a special chisel, such as Krause's.

When an especially accurate reposition of the flap with avoidance of pressure on the dura is desired, a Gigli saw may be passed from one trephine opening to the other and the intervening bone sawed through on a bevel.

A Braatz sound or similar dural separator and protector must be passed between dura and lamina interna, and along this, as a guide, the Gigli saw is passed and manipulated.

We seldom use the Gigli saw method, because of the ever-present danger of damage to bloodvessels and brain in passing the sound and saw and in making the first few strokes. The method also has the disadvantage of being somewhat slow.

When it is desirable to enlarge the bony opening, a new flap may be made with the De Vilbiss forceps, or bone may simply be bitten away in the desired direction with the Macewen rongeur forceps.

Control of Hemorrhage.—Hemorrhage from the bone has now to be controlled, and this is done by (1) pressure made by gauze packed tightly against the bone; (2) packing the oozing bone with Horsley's wax or with muscle tissue from the cut galea; (3) acupuncture with any pointed instrument and mallet. A simple orange stick is cheap, easily sterilized and applied, and is usually at hand; (4) crushing of the bleeding bone with the rongeur forceps is usually successful if other methods fail; (5) for small bleeders we have found the Japanese toothpick very useful.

The best way to avoid hemorrhage from large vessels in the diploë or on the inner surface of the inner table is to take a careful x-ray picture in every case. Then large vessels in the bone can almost always

be seen and therefore avoided later at the operation. This is such an important point that it will be dwelt upon subsequently.

Hemorrhage from the dura and cerebrum occurs only when these are wounded, a rare occurrence with our technique. Occasionally a vein runs from the dura into the lamina interna of the cranial vault, and this may be cut through in the incision, but the resulting hemorrhage is readily controlled by ligating the vein on the dural side.

The dura now lies exposed before our eyes. By palpation with the finger-tips we estimate its tension and by observation whether or not pulsation is present.

When tension is much increased, active pulsation is usually absent (this depends upon the relation of the intracranial tension to the blood pressure).

We must also feel for any increase in local resistance, such as would indicate a tumor or an infiltrate; and for signs of fluctuation, due to intracranial fluid or cyst formation.

Incision in Dura.—The incision in the dura is next made. This may be either cross-shaped or a flap, the pedicle of which is placed either in the direction toward which further exploration is to be done, or in the opposite direction from that of the skin flap. Overlapping of the two flaps on closure of the wound is to be desired, thus preventing a brain prolapse and subsequent fungus cerebri.

When the pia-arachnoid lies exposed, a careful examination is again made. Pulsation is looked for; dilated bloodvessels are duly noted, as an indication of an adjacent tumor; likewise differences in color, prominence and smoothness of the surface. Any local tendency to obliteration of the sulci or flattening of the convolutions is regarded as an important localizing point. Palpation with the finger tips of both hands is done to determine possible spots of increased resistance or fluctuation.

Exploratory puncture is then made into suspected areas and the tissue brought away by the needle is examined macro- and at times microscopically, to determine the presence of fluid, pus, or tumor tissue. The search for brain-tissue cylinders by needle puncture, as recommended by Krause, we have found of not much value, and believe it to be dangerous.

Fenger, of Chicago, some years ago, gave this method a considerable trial and invented a special needle for it, provided on one side with a slot with sharp edges. The needle was inserted into the brain, then turned around, thus cutting out a cylinder of tissue. Fenger finally abandoned the procedure, owing to its dangers and unreliability.

Exploratory Puncture for Pus.—We never make this puncture through the dura but only after the membrane has been opened and the subdural space walled off by packing with several layers of iodoform gauze. When this is done the needle is inserted, and as soon as the pus pocket is located an artery forceps is inserted along the side of the needle into the cavity. The blades of the forceps are then spread as they are withdrawn, and thus a large opening is made, with probably less damage

to nerve fibers and bloodvessels than when a knife-blade is used. The cavity is then irrigated, usually with normal salt solution, and packed with iodoform gauze. During all these manipulations the subdural space is kept well walled off with the iodoform gauze, the outer layers of which are renewed when the irrigation is complete. Drainage to the exterior is then made by a knife-thrust through the galea flap directly over the opening to the abscess cavity, and here the drain, either cigarette, spiral or split-rubber tube, is led out.

Closure of the Wound.—Closure of the wound is done in layers with buried stitches of catgut and superficial stitches of silkworm gut. The dura is brought together and when possible the edges overlapped, unless decompression is aimed at, when it is left unsutured. Sutures are of No. 1 and 2 iodized catgut.

The bone flap is replaced, attached to the galea, but no sutures inserted in it as a rule. The periosteum, unless much stripped up, is left unsutured. Tension stitches of heavy silkworm gut are now inserted into the galea flaps and between them approximation stitches of horsehair or light silkworm gut.

The wound is first washed with bichloride, 1 to 1000, then with alcohol, and finally sealed with tincture of benzoin and silver foil, or less frequently with collodion on narrow gauze strips.

Fluffed gauze is applied over the wound and then a "doughnut roll" of gauze is placed around the wound to protect the bony flap and brain below it from external pressure.

Over the whole bandage a large combination is applied, and lastly a roller bandage held with adhesive strips.

Stimulation of the Patient.—Before the operation the patient's left cubital space is prepared so that an intravenous infusion of normal salt can be given instantaneously, if necessary. A hypodermoclysis apparatus for simultaneous administration of normal salt under both breasts is in readiness during the operation.

A hypodermic of thirty minims of camphorated oil, or $\frac{1}{100}$ gr. digitalin, is given usually before the patient leaves the table, if any evidence of shock or collapse be present, and he is put to bed with his head low. On returning to consciousness the patient is allowed to suck ice, or is given teaspoonfuls of hot water, whichever he prefers.

Normal salt enemata of a pint each are given at intervals of four hours for thirst and for purposes of stimulation.

Continuous rectal administration of normal salt solution I have never found as satisfactory as its administration at intervals, owing to the inconvenience the tube causes the patient and the constant soiling of the bedding.

Postoperative Treatment.—If postanesthetic vomiting is prolonged, the stomach is washed out with a couple of quarts of warm water, which usually gives prompt relief.

When vomiting has ceased, the patient is put on liquid diet, and, if convalescence is normal, a couple of days later on light diet, and at the end of the first week a general diet is allowed.

If there is considerable seepage of cerebrospinal fluid after the operation, the outer dressings are changed as often as saturated, and the inner dressings daily. Any stitches around which necrosis is threatening are removed at the daily dressings and necrotic tissue is promptly clipped away. Tincture of benzoin is applied to the cut surface, and is usually swabbed over the whole wound at every dressing.

When dressings must be frequently changed, they are retained by a large figure-of-eight bandage instead of the roller.

When there is no discharge of the cerebrospinal fluid, the dressings are not removed until the end of the first week, at which time the stitches are taken out, tincture of benzoin swabbed over the wound, and the dressing reapplied for another week, at the end of which time it may be left off entirely. As long as cerebrospinal fluid continues to discharge, careful aseptic dressing must be continued. The use of the silver nitrate stick and tincture of iodine swabs hasten the closing of these fistulæ. Not infrequently they occur through a stitch puncture, especially if the stitch be left in too long. For this reason we often remove stitches as early as the fourth day if the wound is well united, but usually from the fifth to the seventh day.

Occasionally a patient will suffer pressure symptoms when a long open fistula finally closes. In such cases it must be reopened and then again allowed to close gradually.

Patients are usually allowed to sit up on the third or fourth day and leave bed at the end of the first week.

INDICATIONS FOR OPERATION AND DETAILS OF OPERATIVE TECHNIQUE

The indications for operative interference have been roughly outlined previously on page 700. We shall now consider them more in detail and at the same time shall add details to the technique employed for the different indications.

The conditions requiring operation, viewed from a surgical standpoint, fall chiefly into two classes: (1) Focal lesions, localizable and susceptible of radical removal; and (2) lesions causing increased intracranial pressure, and unlocalizable, or otherwise insusceptible of radical removal. We shall consider the latter class first.

INCREASED INTRACRANIAL PRESSURE

This is always a result of a discrepancy in size between the skull and its contents. This discrepancy may be due to a diminution in the size of the brain (most often as a consequence of premature closure of the sutures or "tower-skull"), or to diminution of its capacity by internal apposition of bone (concentric diffuse hyperostosis), or the development of osteomata.

More frequently, however, it is the result of an increase in the volume of the brain, especially from hydrocephalus, tumors and cysts, *granulomata*, abscesses, hemorrhages, edema, or encephalitis.

If the skull be pliable and yielding, the sutures still open and capable of continued bony apposition, and the increase of intracranial contents not too great nor of too sudden origin, a corresponding increase in the size of the skull may take place, so that no active symptoms of increased intracranial pressure ever occur. Congenital hydrocephalus is often a striking example of this compensatory mechanism.

Symptoms.—Headache.—If, however, the skull fails adequately to yield, the symptoms of increased pressure arise. Since they are discussed elsewhere in this work, they will be mentioned here only in passing. Of them *headache* is the most frequent. It is rarely, if ever, entirely absent during the entire course of the disease. A patient may, however, be free from it during considerable periods, particularly if the pressure increases only intermittently. The reference of the headache to particular regions has not much value as a localizing symptom unless combined with sensitiveness to pressure or percussion, or change in note on percussion.

Vomiting.—This is a much less frequent and less valuable symptom than headache. It is present in only about half of the cases, and in only a part of these has it a cerebral character, that is, being unaccompanied by nausea and retching, occurring and increasing with the headache, occurring on an empty stomach, and having a projectile character.

Vertigo.—Vertigo is an uncertain symptom and its chief value is not as a pressure but as a localizing symptom in connection with nystagmus, in which connection it will be discussed later, when speaking of the cerebellum.

Psychic Symptoms.—Psychic Symptoms; clouding of consciousness, stupor, coma, mental deterioration and dementia are late symptoms. Their value in the diagnosis of increased pressure is still further diminished by the frequency of their occurrence in many other conditions.

Generalized Convulsions.—Generalized convulsions do not directly indicate the presence of increased intracranial pressure because they may be due to other causes; but their real value is in pointing directly to the brain as the site of the causative lesion; and when the attacks have a Jacksonian character they point indubitably to the central region as the site of the lesion.

Choked Disk as a Sign of Intracranial Pressure.—The presence of *choked disk* or *optic neuritis* has much more value in the diagnosis of increased intracranial pressure than any of the symptoms yet mentioned. In a previous article,¹ in discussing the nature of optic neuritis and choked disk, we followed Oeller in considering their etiology, pathogenesis, and clinical significance as practically identical. We stated that the difference between them was one of degree only, the choked

¹ In Wood's Ophthalmic Operations, Chicago, 1910.

disk being the more advanced stage, and always first passing through the stage of optic neuritis. Some ophthalmologists have put this limit at 3 D., and others at 4 D., so that it is evident that there is no hard-and-fast line of demarcation. The whole subject is so important, from a surgical standpoint, that we shall discuss it here somewhat at length.

Pathogenesis of Choked Disk.—In spite of the attempts of the advocates of the two different theories, the *mechanical* and the *inflammatory*, to attribute all cases of optic neuritis and choked disk to one common pathogenesis, it is becoming more and more evident that different pathological processes may produce the same macroscopic and microscopic picture in the optic nerve and disk. Since our conception of the pathogenesis is the basis for treatment, we may be permitted to enter into it in some detail.

When v. Graefe first gave the name "*Stauungspapilla*" to this condition, he considered it due to venous congestion in the optic veins, because in some cases, even with hemorrhages present, he found no signs of inflammation in the nerve.

Virchow, on the other hand, found inflammation of the nerve trunk in a case of meningitis with disk changes of only moderate degree, which he assumed to have been communicated to the nerve from the meninges, and to this form v. Graefe gave the name "descending neuritis." Therefore even in v. Graefe's time we find, just as today, a two-fold pathogenesis of optic neuritis.

The "descending neuritis" was supposed to be characterized ophthalmoscopically by only a slight degree of swelling in the disk and a tendency to invade the retina; while the cases associated with increased intracranial pressure, *e. g.*, tumor, were characterized ophthalmoscopically by a high degree of swelling limited to the disk, the retina remaining more or less unaffected.

We now know that these ophthalmoscopic differences, as given above, are artificial, and that cases associated with high intracranial pressure may present only a slight disk change, or none at all, while meningitis, pyemia, intoxications and other conditions associated with a "descending neuritis," and without other evidence of increased intracranial pressure, may present the typical appearance of choked disk, so that we no longer attempt to make an ophthalmoscopic, etiologic diagnosis between "descending neuritis" and choked disk.

v. Graefe considered that choked disk was produced by the increased intracranial pressure damming back the circulation of the eye by pressing on the cavernous sinus. He thought that this mechanical effect was still further enhanced by the effect of the rigid scleral foramen.

This theory of pressure on the cavernous sinus received a severe setback, in 1869, when Seseman showed that the communication between the orbital and facial veins is so free that pressure on, and even obliteration of, the cavernous sinus produces no intra-ocular changes. This was further corroborated by Hutchinson's case, in which complete obliteration of the cavernous sinus by pressure from aneurysm produced no eye changes.

It was Schwalbe, in 1869, who first showed that the subvaginal space around the optic nerve is continuous with and can be injected from the subdural space, and who pointed out the significance of the fact in connection with choked disk.

Carion, Manz, and Schmidt showed how frequent is the distention of the optic sheath in neuritis, and tried to produce optic neuritis by injecting the subdural space with fluid; and in some cases a transient swelling and redness of the disk resulted, but no typical neuritis.

Schmidt found that fluid injected into the sheath passed into the lymph spaces in the nerve at the *lamina cribrosa*, and he suggested that pressure *inside* the nerve and perhaps irritation from the fluid in the nerve causes the neuritis.

Schmidt's and Manz's views were widely accepted in Germany.

Leber threw his influence in favor of the inflammatory theory, in 1881, in a discussion at the International Medical Congress in London, and here first used the term "papillitis." He rejected the theory of Manz that the fluid acts by mechanical pressure, and also failed to verify the connection between the sheath and lymph spaces of the nerve, asserted by Schmidt-Rimpler. He assumed that the fluid in the sheath excited neuritis by conveying pathological material into the optic nerve behind the eye. Deutschmann published additional evidence in favor of Leber's theory.

Other theories of the origin of optic neuritis which have arisen only to be discarded are: (1) the "foreign body" theory of Hughlings Jackson and Brown-Séquard; (2) vasomotor theory of Benedikt; (3) inflammation by continuity theory of Galezowski; (4) basal meningitis theory of Edmunds and Lawford; (5) theory that neuritis optica intra-ocularis is the expression of a general cerebral edema, the effect of distention of the ventricles of the brain.

In more recent years the Schmidt-Manz mechanical theory has been upheld by Schulten, Bramwell, Oppenheim, Hoche, Merz, as well as v. Bruns and Cushing. Uhthoff, Saenger, Axenfeld, and Kampfferstein speak of rigid *lamina cribrosa*, in addition to increased intracranial pressure, as furthering intra-ocular passive congestion.

The Leber-Deutschmann theory has been vigorously upheld by Knies and Gowers, who believe in a descending neuritis; the same opinion is also held by Elschmig, who first used the term *papilledema*. Jacobson and Fleming have likewise upheld the Leber-Deutschmann hypothesis.

But whether the process in the nerve and nerve head is inflammatory in nature (as evidenced by round-cell infiltrations, exudate, edema, hemorrhages, increase of nuclei), or merely a passive congestion and edema, it is certain that in the great majority of cases, and particularly in those in which an increase in intracranial pressure is demonstrable, decompressive operations lead to retrogression of the eye conditions, and even to the complete or almost complete recovery of vision in eyes previously nearly, though rarely quite, blind.

The time of election for operation in these cases is the beginning of

loss of vision. Later operation, when atrophy is advanced, cannot restore the damaged nerve cells.

One important reason for the present confusion in the entire subject of choked disk and optic neuritis is the lack of an adequate terminology. We apply these terms to designate something which we see in the fundus by the ophthalmoscope; and instead of using terms of a purely objective descriptive character, we use terms which refer to the supposed pathogenesis of the lesions and not at all to the changes which we actually observe. Therefore, four different observers may call the same lesion optic neuritis, choked disk, papillitis, or papilledema, and yet all these four terms imply rather distinctly different processes. The changes which can be observed in the fundus in such cases are actual facts in the description of which all observers can agree. These actual facts should be sharply separated from the theories regarding pathogenesis, a matter in which there will continue to be much difference of opinion.

Another reason for the existing confusion is that these fundus changes are not always observed from their beginning to their end by the same medical man, or even by *any* medical man. What we see as a choked disk of five diopters may have started as a pure papilledema, and later have developed hemorrhages and exudate, or it may have had them from the first, or it may run its entire course without them. To observe the beginning of these lesions seems at present to be especially important. To this end every practitioner and surgeon should be urged to learn to use the ophthalmoscope. The electrical ophthalmoscope has now become a popular instrument, which every medical man should own and use as frequently as he does the stethoscope. The ingenious new electric ophthalmoscope recently devised by my associate, Dr. Roger T. Vaughan, a description of which was published by him in the *Munch. med. Woch.* for 1913, reduces the electric ophthalmoscope to pocket size, and lowers its price to within the reach of every practitioner who has the ambition to learn its use. The findings of the ophthalmoscope are not a whit less important nor its mastering more difficult, at least in the case of the electric instrument, than the stethoscope. As Cushing insists, every man who does neurological surgery should either be able to use the ophthalmoscope reliably himself or have an assistant who can. If the ophthalmoscope were in much more general use, and if we had a better array of purely objective designations for the nerve-head pictures which we see, we should make much more progress in the clarifying of the subject of choked disk. Just at present the mechanical theory seems to have the upper hand, and the papilledema of Elschnig, a transparent swelling of the disk without hemorrhages or exudate, appears to be the type which, while not very frequent, speaks, when found, most strongly for the existence of increased intracranial pressure.

Etiology of Choked Disk and Optic Neuritis.—The following conditions are those in which neuritis intraocularis and choked disk more commonly occur.

A. INTRACRANIAL CONDITIONS.—*Tumor Cerebri*.—1. Tumor cerebri is by far the most frequent cause of choked disk. In 200 cases of Kampfferstein's of choked disk, 134 were due to brain tumor. Had cerebral syphilis and conglomerate tubercles been added, the number would have been raised to 170.

Gowers holds that tumor cerebri is also the most frequent cause of optic neuritis, though R. Greff is inclined to give the precedence to the various forms of meningitis.

The size of the tumor plays no great role. Choked disk may occur with very small tumors or be absent with very large ones. Much more important is the location of the tumor, and whether it interferes with the drainage of the ventricles or the sinuses, especially the sinus rectus, and the veins of Galen.

The location of the tumor is of much more importance in the production of choked disk than its microscopic character or specific etiology. In general it may be said that the tendency of tumors to produce choked disk increases from the anterior to the posterior pole of the brain, and diminishes from the cortex inward. Also choked disk is somewhat more likely to occur earlier and reach a higher degree on the same side as the tumor.

Paton, in reviewing 252 cases of brain tumor, found that tumors of the cortex are more liable to produce optic neuritis and choked disk than tumors of the subcortical white matter and pons, and that the intensity of the neuritis in tumors of the cerebral cortex varies inversely with the distance of the part affected from the anterior end of the middle fossa.

Age is an important factor in the production of choked disk by tumor, youth being much more liable to develop it than age. H. D. Singer, in reporting 88 cases of brain tumor, showed that the average age of 51 cases with well-developed choked disk was twenty-eight years, and of 9 cases without choked disk was fifty-four years.

Heredity, sex, nationality, and occupation play little if any role.

Tumors of the posterior fossa are especially liable to produce choked disk. They are: cerebellar cysts, psammomata, endotheliomata, sarcomata, gliomata. The cerebellum is also the location of predilection for conglomerate tubercles and otitic abscesses.

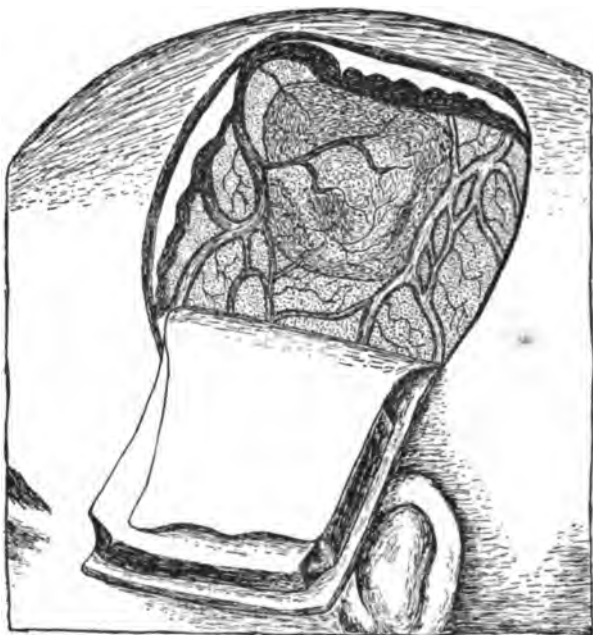
Cerebellopontine angle tumors most often grow from the acoustic nerve; less often from the adjacent nerves, corpora quadrigemina or cerebellum. Acoustic tumors are usually fibromata, gliomata, sarcomata, and less often psammomata, and not rarely may be bilateral.

Basal tumors and hemorrhages (also of the corpora quadrigemina, pons, medulla, pineal gland, etc.) produce choked disk much less often than the two groups above, perhaps because, owing to early involvement of vital parts, they produce death before they arrive at the choked-disk-producing stage.

Hypophysis tumors and *cysts* (occasionally gummata and rarely tuberculomata) may sometimes produce choked disk, but set up optic neuritis somewhat oftener.

The characteristic eye finding of hypophysis tumor is bitemporal hemianopsia. Binasal hemianopsia is rarely found; bitemporal atrophy, atrophy in varying grades of one or both eyes, or hemianopsia with atrophy are somewhat less characteristic. They usually find an adequate explanation at *postmortems* in direct pressure of the tumor on the optic tracts or chiasm, and less frequently on the optic nerves themselves, to reach which a pituitary tumor must be very large and must have a very unusual forward extent.

FIG. 73



Subcortical tumor. The dilated veins and discolored, uneven cortex enable us to locate such subcortical tumors at operation. Any one of these three characteristics is enough to act as guide for an exploratory incision.

Erdheim, in 1904, showed and depicted the mechanism by which both optic tracts may be caught between a growing hypophysis tumor and the carotid arteries, and crushed between them. Erdheim, in 1909, had another similar case, an unsuccessful attempt at extirpation by Hochenegg, with death from hemorrhage, and in this case the two very rigid sclerotic carotids acted as unyielding bands, between which and the large pituitary tumor, the optic tracts had been compressed to a thin, scarcely perceptible transparent ribbon. The patient, needless to say, was completely blind before the operation.

Perhaps the very rare cases of pituitary tumor with binasal hemianopsia are due to such a condition, the pressure of the carotids on the temporal fibers of the tract making itself felt before the pituitary tumor itself has brought about a distinct lesion in the nasal fibers.

From the mechanical theory of optic tract lesions in hypophysis tumors, Schnabel seems to be the chief dissenter. His theory, brought forth in 1906, that the bitemporal hemianopsia is of toxic origin, due to the direct action of pituitary toxin on the nerve fibers, and likening the condition to toxic central scotomata, has not found much support. Physiological and chemical researches since that time, while they have considerably enlarged our knowledge of the pituitary secretion and its functions, have not as yet demonstrated a neurotoxin sufficiently active to produce, unassisted, so severe a nerve lesion as that occurring commonly with pituitary tumor. Moreover, since the appearance of Schnabel's article, the relief experienced from operative procedure in patients, in most of whom only a partial extirpation of the gland has been performed, speaks rather strongly against the toxin, and for the pressure theory.

Cortical tumors (those above the tentorium) do not usually develop neuritis, though they do so more often than tumors of the subcortical white matter. From the point of view of diagnosis and treatment, these tumors should be divided into those of the (1) sensorimotor area; (2) occipital lobe; (3) temporal lobe; (4) frontal lobe; and also those arising from the (1) meninges; (2) outside the meninges (in the bone and outer surface of the dura); (3) cortex; (4) subcortical white matter and spreading to the cortex.

Cerebral cysts almost deserve a chapter to themselves, owing to their peculiar, varied, and often obscure etiology, and to the problems which they present in diagnosis and treatment.

They are: (1) primarily cystic tumors; (2) solid tumors with secondary cystic degeneration; (3) traumatic cysts (a) hemorrhage with subsequent absorption; (b) encephalomalacia, etc.; (4) connective-tissue cysts of Krause; (5) parasitic; (6) lymphatic.

2. *Brain Abscess*.—Brain abscess is, according to Oppenheim, who has reviewed the literature up to 1909, in 30 to 50 per cent. of the cases:

(a) *Of otitic origin*. These otitic abscesses are located either (1) extradurally, (2) in the cerebellum, or (3) in the temporal lobe. They are mentioned here in the order of their frequency.

(b) *Disease of the accessory sinuses* produces brain abscess much less often than does ear disease. The location of these abscesses is usually either subdural at the base or in the frontal lobes.

(c) *Abscess following injury* may be located anywhere, depending on the site and nature of the trauma.

(d) *Pyemic and metastatic abscesses* may occur anywhere and do not show the same uniform tendency to attack the region of the basal ganglia that embolic hemorrhages show. We may also mention *brain abscesses occurring with bronchiectases and other purulent affections of the lungs*. The route of infection of the brain here is not always entirely clear.

3. *Intracranial Hemorrhages*.—These are much less frequently the cause of choked disk than the above-mentioned conditions. In fact, in the great majority of cases, even with massive hemorrhages into

the meningeal spaces, there are no changes in the eye-grounds. Why swelling of the disk is present in the small minority of cases and absent in the others is difficult at present to state, but it seems in line with the relatively small tendency of cortical disease in general to produce choked disk, and with the fact that the hemorrhage must cease as soon as the intravascular and intracranial pressures become equal. In the fatal cases complicated by meningitis, swelling of the disks is somewhat more frequent.

4. *Sinus Thrombosis*.—Sinus thrombosis in two-thirds of the cases produces no eye changes; why in some cases and not in others is difficult to say. It cannot be a matter of restriction of the outflow from the ophthalmic veins, since many cases of complete blocking or thrombosis of the cavernous sinus, as first reported by Hughlings Jackson, show no fundus changes, the blood escaping freely from the ophthalmic veins by way of the facial and angular veins and their tributaries. Very likely some cases run their course with increased intracranial pressure while others do not.

5. *Meningitis*.—Meningitis is a very common cause of optic neuritis (*i. e.*, of a moderate degree of papillitis), but rarely of actual choked disk.

Epidemic meningitis is a frequent cause of optic neuritis, of which it is often one of the earliest and most valuable signs, as demonstrated by a case seen by the authors at Cook County Hospital in January, 1910, in which an examination-room diagnosis of lumbago was changed to one of epidemic meningitis by the finding of a bilateral optic neuritis. This led to a lumbar puncture, resulting in finding pus and the diplococcus intracellularis.

Choked disk, when it does occur in meningitis, is apt to appear late in the disease, when the acute symptoms have disappeared. A remnant of exudate, more or less organized, blocks the foramina of exit of the fourth ventricle (foramina of Magendie, Key, and Retzius), and causes an internal hydrocephalus.

Tuberculous meningitis leads to optic neuritis only in rare cases. Tubercles in the choroid are almost as frequent here as is optic neuritis. Choked disk occurs chiefly when large conglomerate tubercles, particularly in the cerebellum or pons, complicate the meningeal disease.

Meningitis from other causes (*pneumococcus, streptococcus, influenza and pyogenic cocci*) leads to optic neuritis in a percentage of cases lying between the above two forms. Choked disk occurs chiefly when they are accompanied by brain abscess, internal hydrocephalus, or pyocephalus.

6. *Hydrocephalus Internus*.—Hydrocephalus internus, particularly in the chronic form, is a frequent cause of the higher degree of optic neuritis, choked disk, and postneuritic atrophy.

Whether choked disk in these cases be due to hydrops of the sheath of the optic nerve, edema of the nerve itself, or to pressure of the dilated third ventricle on the chiasm is not certain. At any rate, it seems likely that the last occurrence may explain the sudden, transient, and recurring

amblyopias which are an alarming symptom in some brain tumors associated with internal hydrocephalus.

7. *Aneurysm of Cranial Arteries*.—Aneurysm of the cranial arteries, most usually at the base (carotids, vertebral, basilar, circle of Willis, antero-venous aneurysm, etc.), is one of the rarer causes of choked disk.

8. *Cranial Deformity*.—Cranial deformity particularly the "*tower skull*," which is often associated with an internal hydrocephalus, is a more frequent cause of the swelling of the disk than *exostoses*, which occasionally, though rarely, present the only demonstrable cause for an existing optic neuritis, which may disappear on their removal.

9. *Brain Syphilis*.—Brain syphilis produces choked disk chiefly in the gummatous stage, or when associated with hydrocephalus. Optic neuritis is, however, common, though often unilateral, in the secondary stage, occurring especially along with the early relapsing eruptions, *rupia*, *condylomata*, *iritis*, etc., rather than with the primary eruption.

10. *Ear Disease*.—It is chiefly the intracranial complications which lead to optic neuritis, especially abscesses, suppurative meningitis, meningitis serosa, and sinus thrombosis. In a certain, though small, number of apparently uncomplicated cases of otitis media there may develop an optic neuritis. It has, however, been noted that later these cases are more apt to present intracranial complications than are those without the accompanying neuritis. Ménière's disease may also occasionally be accompanied by an optic neuritis, perhaps due to the same cause which produces the trouble in the inner ear or vestibular tract.

11. *Encephalitis*.—Encephalitis producing any eye symptoms causes optic neuritis of low grade, and very rarely, if ever, choked disk, unless abscess formation intervenes in addition. Encephalitis occurs with acute poliomyelitis often as an accompanying acute polioencephalitis (Strümpell), scarlet fever and the other exanthems, meningoencephalitis and trauma.

12. *Postoperative Neuritis*.—Postoperative neuritis, as pointed out by Cushing and by de Schweinitz and Holloway, though a rare complication of exploratory and decompressive measures, may occur in eyes previously normal. The more usual occurrence, however, is that a previously existing optic neuritis or choked disk, for a few days or a couple of weeks after the operation, shows a slight or moderate increase in intensity before it gradually disappears or goes over into a secondary atrophy.

B. OTHER LESS IMPORTANT OPTIC CONDITIONS, causing optic neuritis, which, however, may be mistaken for those demanding surgical intervention, are: multiple sclerosis, parietic dementia, nephritis (neuritis or neuroretinitis), neuritis of pregnancy and the puerperium, neuroretinitis leukemica.

Intoxications, especially with metallic salts, are more apt to produce retrobulbar neuritis than neuritis optica intraocularis, and also are

very apt to lead to optic atrophy, *e. g.*, arsenic, lead, mercury, alcohol (ethyl and methyl), tobacco, etc.

Lightning stroke is said to be a cause of optic neuritis, but the so-called "*Berlinische Trübung*" is a more frequent fundus change.

C. ORBITAL DISEASE is a frequent cause of optic neuritis and choked disk, which is, of course, with rare exceptions, unilateral here.

The particular varieties of orbital disease causing swelling of the optic disk, especially those secondary to inflammations or tumors of the accessory sinuses of the nose, are not *apropos* in this work, and will, therefore, not be discussed here.

Diagnosis.—Reversal of the Color Fields.—Bordley and Cushing have described as pathognomonic, for increased intracranial pressure, a reversal of the fields of red and blue, the field of blue being smaller than that for red, while the field for white remains unchanged. After decompressive trepanation, Cushing saw the fields return to normal. Cushing found this as an early symptom in many cases of brain tumor, and even performed decompressive trepanation from this indication alone. This discovery of Cushing's, if true, is very important. It has had, unfortunately, not yet sufficient attention paid to it. So far as we know, no serious attempt has been made to verify or contradict it. On our side of the water the oculists have hastened to remind us that this color reversal is a frequent finding in hysteria, as has long been known. Krause, in his voluminous work on the *Surgery of the Brain and Cord*, mentions Cushing's publication, but says nothing as to his own experience. Our own experience is not sufficient to have much value in this connection, since our material consists chiefly of cases referred by neurologists after the diagnosis has been thoroughly established.

The whole subject of anomalies of the color sense has been investigated lately with typical German thoroughness by Hans Koellner, assistant in the eye clinic at the University of Berlin. He found blue and violet blindness in a number of affections of the optic nerve, as one of the earliest symptoms to appear, and latest to disappear. Especially frequent was it in the different forms of neuritis, particularly of the luetic, albuminuric and sympathetic forms. In ten cases of luetic neuritis it was present six times. He also found it present in choked disk. The blue and violet central or paracentral scotomata were the most frequent forms of the affection. The exact frequency of the peripheral diminution of the perception of blue and violet he does not state explicitly, and appears to be ignorant of Cushing's work, or at least makes no mention of it. That optic neuritis is more frequently accompanied by a progressive red and green blindness beginning peripherally he lays considerable stress on, and also that this is often the earliest sign of the trouble.

Koellner has attacked the whole subject of the color sense disturbances in a very thorough-going and fundamental way, and his book represents many years of laborious examinations and constitutes the most important advance in this field perhaps since Helmholtz's time.

Instead of following the simple perimetric method of Cushing, henceforth, all those who wish to do further research along this line must be prepared to make complete examinations of the entire color sense in these cases, including the use of pseudo-isochromatic tables, the Rayleigh comparison test, the anomaloscope, etc. Highly desirable as such examinations are, both from a scientific and practical standpoint, it seems scarcely likely that we shall see them made extensively in American clinics for a long time to come. It is difficult enough to get competent assistants to make adequate perimeter charts without expecting, in addition, the large amount of training and leisure time requisite for careful complete color tests of the entire field. In view of Koellner's experience, it seems at best very improbable that Cushing's syndrome is at all pathognomonic for intracranial tumors; but that it is rather an early symptom in the different forms of neuritis, less frequent in choked disk, and may also be found in other intra-ocular affections, optic atrophy, albuminuric retinitis, etc.

Bradycardia.—Bradycardia is an important point in the diagnosis of increased intracranial pressure when present. The pulse beat may sink to forty per minute, or even less, usually accompanied by well-filled arteries and an increased blood pressure. Unfortunately, it is chiefly in the acute and not in the chronic cases that it is found.

Depression and Paralysis of the Respiration Centre.—This is seen chiefly late in the course of brain tumor, and is due to pressure on the medulla. It may lead to death before there are any localizing symptoms of the tumor present. This type of death is one of the common ones after intracranial operations, even following so slight an operation as brain puncture. The rapidly increasing pressure in such cases is probably due to an acute edema of the brain.

The X-ray.—We come lastly to one of the most important signs of increased intracranial pressure, the only pathognomonic one which we possess. It is the presence of generalized erosions of the calvarium and increased formation of jugal cerebralia at the base. When present, we can say at once, without further evidence, and even in the absence of clinical symptoms, that there is or has been recently, increased intracranial pressure. Dr. Arthur Schüller, of Vienna, saw and interpreted these changes and published them in 1905, in his monograph. Why, in the intervening seven years, the frequency and the significance of this finding has not come to be more generally understood is hard to say. In this country Spiller, of recent years, has written about the importance of the x-ray examination of the skull; but the bulk of the profession has not had its attention called particularly to the importance of these changes. There is at present no text-book in English which includes the x-ray picture as one of the cardinal points in the diagnosis of increased intracranial pressure and intracranial growths. Besides the positive diagnosis of increased intracranial pressure which can be made from an x-ray of the skull, the other important points in the diagnosis which may be made with its help are:

1. *Craniostenosis* (of which "tower skull" is the most important form).—This is due to premature closure of the sutures of the skull, the form of skull varying according to the sutures obliterated. (Scaphocephalus, brachycephalus, dolichocephalus, etc.)

2. *Enlargement of the Sella Turcica*.—This may be due to tumor of the hypophysis, dilatation of the third ventricle with general or localized hydrocephalus, or tumor of the base of the skull extending to the sella.

3. *Internal exostoses and concentric hypertrophy of the skull*.

4. *Calcification of the Brain*.—This is most often seen in the pineal gland, which is located in the x-ray picture one and one-half centimeters behind and three and a half centimeters above the external auditory meatus, and in the midline of the body. When the pineal gland is thus visible, its lateral displacement may sometimes be used in determining the side of the brain in which the new growth is located.

5. *Veins in the Diploë and Internal Table*.—When there is an obstruction to the venous flow in the sinuses, the building of a collateral circulation in the bone will sometimes give a clue to the location of the blocking, just as does the formation of the caput medusæ in cirrhosis of the liver (*e. g.*, a recent case of ours of acusticus tumor).

6. *Epithelioma of the Dura*.—Diagnosis of the localization of epithelioma of the dura. Here a hyperostosis sometimes occurs in the inner table just over the tumor. Infiltration of the bone by the tumor is also not infrequent, and even penetration to the outside may occur, as we have twice seen.

7. *Erosion of the Bone*.—Erosion of the bone from disease processes penetrating from the inside to the outside, or *vice versa*, especially tumors, tuberculosis, and syphilis.

For the many other rarer pathological processes in which the x-ray is more or less a factor in the diagnosis, we must refer those interested especially to Schüller's recent work in the Nothnagel series, and also to his chapter in Lewandowsky's *Handbuch*.

Nystagmus.—Another important point in the diagnosis of intracranial processes, particularly in the septic processes going out from the ear, and in the diseases of the cerebellum, is the examination of the character of the nystagmus as developed by the Vienna school of otology, and by Barany in particular. This subject, like that of the x-ray of the skull, requires a monograph of its own for its adequate and understandable presentation. It cannot be properly handled in the small space at our disposal. We shall, therefore, cite chiefly the literature in which it can be best studied; and shall refer only to certain of its findings later, particularly when we discuss the treatment of diseases of the cerebellum.

Localization.—The localizing symptoms of the different intracranial affections are not presumed to be discussed in this work, and therefore, will be considered in this chapter only in passing, as the procedures for their operative relief are set down. We can add here that every

intracranial condition demanding operative interference should have not only a thorough general physical and neurological examination, but should have eyes, ears, nose, pharynx, and larynx examined. The Hay pharyngoscope we can especially recommend for obtaining a good view of the retronasal space, from which tumors may grow into the base of the brain and into which tumors of the base may penetrate; for instance, large hypophysial tumors, chordoma as in Spiess' case, epithelioma of the dura at the base, sarcoma and carcinoma of the nose, sinuses, pharynx and tonsils, and the like. For the easy examination and diagnosis of such tumors, Hay's instrument marks a considerable advance over the old retronasal mirror.

Serological Methods for the Diagnosis of Malignant Tumors.—Before dismissing the subject of the diagnosis of intracranial tumors, it may be worth while to mention the recent attempts, serological and otherwise, to diagnosticate occult malignant tumors. These attempts are more significant for the hopes they have raised than for the results they have so far achieved.

The oldest and the most reliable as yet of them all seems to be the *meiostagmin reaction of Ascoli*. The basis for this reaction is the observation that when an antigen and antibody are brought together there occurs a decrease in the surface tension of the mixture, and therefore an increase in the number of drops in a given quantity of the mixture. According to this decrease in the surface tension, as measured by the Traube stalagmometer, the reaction is called positive, doubtful, or negative.

Freund and Kaminer, in Vienna, have found that the serum of carcinoma patients has no cytolytic action on carcinoma cells, whereas normal serum dissolves them. This fact they made the basis for their test. Unfortunately, only about 70 per cent. of carcinoma sera give the reaction, and some normal sera do as well.

E. von Dungern still more recently has applied the complement fixation method, using as his antigen the tumor extract in 98 per cent. alcohol, which is therefore a lipoid emulsion. The malignant tumors give the most marked reaction, but benign tumors also give a positive reaction, though less markedly so. All other cases so far examined give a negative reaction. In some cases it is advisable to try several antigens if the test is not positive with the first one.

Salomon and Saxl, from Van Noorden's clinic in Vienna, have reported a new sulphur body in the urine which they consider nearly, although not quite, specific for carcinoma. This body is split off by hydrogen peroxide and is demonstrated as a precipitate with barium sulphate. The administration of antipyrine and creosote interferes with the reaction.

R. Kraus, Graff, and E. Ranzi have recently tested out these different reactions and give the preference to the meiostagmin reaction, although admitting that all these reactions are as yet far from being entirely satisfactory.

Von Monakow has come to the same conclusion.

E. E. Pribram finds Salomon and Saxl's sulphur reaction not specific, but positive in at least sixty per cent. of the carcinoma cases.

The Treatment of Increased Intracranial Pressure.—This is essentially an operative one. There are no medicinal or palliative measures of any importance. The only proceeding of any value which does not lie strictly in the province of the surgeon is lumbar puncture. To this measure we shall refer later. The choice of the other procedures depend upon the character and location of the lesion causing the increase in intracranial pressure, and the possibility of its radical removal. We shall simply enumerate here the operations which are performed for the relief of increased intracranial pressure, and shall leave the discussion of their technique and limitations to a later part of this article.

1. **The Subtemporal Decompression of Cushing.**—(For technique, see page 724.) This operation is especially valuable in cases of unlocalized growing tumors, especially when above the tentorium. It has also been recommended together with brain puncture or Payr's drainage of the ventricles for internal hydrocephalus. The advantage of this location for decompressive trepanation is that the bulging brain is protected by the temporal muscle, which allows it gradually to expand without forming a large hernia, and prevents the development of blindness while waiting for localizing symptoms to appear. The decompression is usually done on the right side in a right-handed person, and is therefore over a silent area, save that occasionally, if the prolapse be large, the lower Rolandic area may be involved in it. If done on the left side, it would involve the temporal convolutions with consequent danger of the production of aphasia. It is possible that it may occasionally have the same result on the right side, in right-handed persons, now that we know that occasional centres are on the right side in right-handed persons, as has been shown by Kurt Mendel and others.

2. **Suboccipital Decompression.**—This operation is chiefly valuable in cases of tumor below the tentorium, unlocalizable but growing. Since the space below the tentorium is thereby relatively accessible to the surgeon, it is usual in most cases to do a thorough exploration at the same time, and only in case this examination fails to reveal the tumor, or in case it shows the tumor to be inoperable, is the operation turned into a decompression. Puncture of the fourth ventricle for hydrocephalus or for cysts may also be combined with this operation. The prolapse in such cases is not quite so well protected as in the cases of subtemporal decompression; but still the nuchal muscles cover it fairly well and the hair soon hides the bulging from view.

3. **Sellar Decompression.**—A. E. Halstead's technique for opening the sella turcica will be discussed later in connection with the other methods of its approach. Since it is relatively easy with the x-rays to diagnosticate those cases of internal hydrocephalus accompanied by marked enlargement of the third ventricle and consequent expansion of the sella turcica, Arthur Schueller has recommended the operative performance of what occurs spontaneously in some of these cases, the puncture of the third ventricle by way of the nose and sella turcica,

and the formation of a permanent fistula into the nose or pharynx. He recommends a simple needle puncture by Hirsch's submucous intranasal route, but as yet has had no cases of his own. One of the fatalities in Hirsch's series of hypophysis operation was the result of thrusting a knife into such a dilated third ventricle, which he had wrongly diagnosticated as tumor of the hypophysis. The patient died in a few hours from the loss of spinal fluid and intracranial hemorrhages, consequent to diminution of the intracranial pressure following this loss of fluid. Had Schueller's method of needle puncture been followed in this case, not only might the patient have been saved, but perhaps permanently relieved as well. Anton has condemned this procedure because of the danger of septic contamination from the nose, and has preferred his "Balkenstich" to it. Until this operation has been actually performed in a number of cases it will be impossible to say positively much about it. On a theoretical basis it has much in its favor, namely:

- (a) Simplicity—a simple needle puncture under novocaine.
- (b) Draining the ventricles at their lowest point, and when there is a tendency spontaneously to perforate.
- (c) Probable formation of a permanent fistula, with permanent relief of the pressure.

This procedure aside from lumbar puncture is the simplest one which has been proposed for the relief of internal hydrocephalus. It deserves to be tried before being condemned as unclean. Hirsch's record of twelve hypophysectomies by this route without an infection may well lead one to doubt the justification of Anton's criticism as to its uncleanliness.

4. Atypical Decompression.—In not a few cases an operation undertaken for the removal of a localized tumor fails to reveal a tumor, and the operation has to be turned into a decompression. These decompressions, done directly over the tumor, which is often growing more or less rapidly, and which is thereafter covered only by galea and skin, are those which are most likely to give rise to prolapse and fungus cerebri. If the tumor be rapidly growing, this tendency to the production of prolapse may be somewhat controlled by making a second decompression at one of the submuscular or other available sites. The various other precautions to prevent the occurrence of prolapse in such cases will be discussed later (page 749).

In traumatic cases with fracture of the skull, hematoma formation or laceration of the brain itself, an atypical decompression must be done; but in such cases there is rarely any danger of prolapse, since if the hemorrhage be stopped there is no further increase in the intracranial pressure. In genuine epilepsy both Kocher and Krause recommend in some cases a simple decompression over the primary epileptic centre, if known; if not, then at one of the submuscular sites. In these cases, as a rule, there is no danger of prolapse, because the intracranial pressure, if at all increased, is only slightly so.

5. Puncture of the Corpus Callosum.—The object of this procedure is to make a connection between the ventricles of the brain and the

subdural space, so that in case of internal hydrocephalus the fluid may find its way into the latter space. Anton recommends this procedure not only in congenital hydrocephalus, but also in those forms secondary to tumor, and having choked disk; also with the pseudotumor and in some of the forms of epilepsy. Its advantages are cessation of the headache, improvement of the vision, and retardation of the amaurosis. It also has the advantage of eliminating the symptoms due to accompanying hydrocephalus and, therefore, aids in the ultimate localization of the tumor. Permanent drainage of the ventricles by this method is, however, only possible when the subarachnoid space can absorb the cerebrospinal fluid; and this is not always the case.

6. **Brain Puncture.**—Brain puncture is a much older procedure than the foregoing, having been done at intervals since the middle of the last century for the relief of internal hydrocephalus. It affords only a temporary relief, however, of the intracranial pressure, and must therefore be frequently repeated to afford relief. In this respect it is therefore inferior to the methods of Anton and Schueller.

7. **Permanent Drainage of the Lateral Ventricle.**—Nicholas Senn and later Payr and others conceived the idea of following brain puncture with the insertion of a permanent drainage-tube through the opening. Instead of a tube of rubber, gold, or silver, ox arteries have been used and also folded suture material, catgut, and the like. Thus the establishment of permanent drainage between the ventricle and the subarachnoid space is aimed at. The results so far, however, have not been generally satisfactory.

8. **Spinal Puncture.**—The use of lumbar puncture was in great vogue among internists and neurologists for the relief of chronic internal hydrocephalus and other forms of increased intracranial pressure until Cushing pointed out the danger—especially when the cause of the increased intracranial pressure lay above the tentorium—of causing hernia of the medulla into the foramen magnum, with consequent paralysis of heart and respiration. Cushing was thus the first to give a logical explanation of the not infrequent occurrence of sudden death following this procedure. In this class of cases lumbar puncture is now no longer in good repute, and should be discarded in favor of one of the other operative methods.

9. **Permanent Drainage of the Spinal Subdural Space.**—(a) Into the subcutaneous tissue of the back.

(b) Into the peritoneal cavity by perforation of the body of a vertebra after laparotomy (Cushing and McArthur).

In cases of chronic internal hydrocephalus and other conditions causing increased pressure above the tentorium, these procedures are open to the same objections as spinal puncture. These spinal procedures represent a more extensive and serious undertaking than the intracranial operations, and do not attack the problem so directly as the latter. They are mentioned in this connection only to condemn them.

Of all these procedures, at present available for the relief of increased intracranial pressure, the most generally available is the decompression

flap operation. The simpler operations for the relief of internal hydrocephalus are still in too experimental a stage to permit of a definite opinion as to their efficiency being formed. The methods of Schueller, Anton, and the permanent drainage of the lateral ventricle by tube or wick seem to present the most promising outlook.

OPERATIVE PROCEDURES FOR SPECIAL INDICATIONS IN TUMOR CEREBRI

The general technique of exploration and decompression for cerebral tumor has been already described. It remains now to speak of (a) *subtemporal decompression* (H. Cushing) for unlocalizable or inoperable tumors above the tentorium; (b) *suboccipital decompression*, usually bilateral, less often unilateral, for exploration and decompression in the case of disease located below the tentorium; and (c) *special procedures* practised in the removal of certain tumors.

The tumors are (1) cysts, (2) tubercles, (3) vascular tumors, (4) cortical and subcortical tumors, (5) encapsulated tumors, and (6) hypophysial tumors.

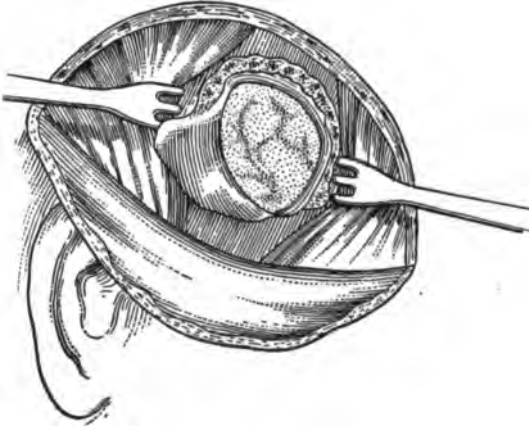
It also remains to give briefly the technique of (a) hemostasis, (b) obliteration of the resulting cavity after tumor excision, (c) exploratory incision of the cortex, (d) the two-stage operation, and (e) the prevention of shock and collapse.

(a) **Subtemporal Decompression.** — Subtemporal decompression, done that the temporal muscle may reinforce the skin and galea in restraining postoperative *prolapsus cerebri* after decompressive procedures, thereby avoiding a fungus, with consequent dangers of septic infection of the brain and meninges. In our clinic a semicircular incision is made parallel to and one centimeter below the origin of the temporal muscle. Anteriorly, it reaches not quite to the hair border and, posteriorly, it runs somewhat lower, behind the pinna of the ear. Skin and superficial fascia are stripped downward toward the base of the flap, leaving the temporal muscle and fascia exposed. These are now separated bluntly in the direction of their fibers down to the periosteum. The two edges of the muscle are also separated to a maximum extent by blunt retractors. The periosteum is then incised and pushed back with an elevator on both sides as far as possible. Then the cranial cavity is opened in the usual way, except that the bone is bitten away with the Macewen forceps over the entire area of the decompression. A dural flap is made with the base upward, opposite the base of the skin flap. The decompression is thereby completed and it remains to close the wound. Catgut sutures unite the edges of the temporal muscle and fascia. The skin is sutured with silkworm gut. The usual dressing is then applied and the after-treatment is the same as for the decompressive measures previously mentioned.

We have had very good results with the Cushing technique in so far as the prevention of prolapse is concerned. Occasionally it has

not been possible to approximate the muscle edges closely, owing to the bulging of the brain. In this case we close the opening in the muscle as well as possible below and trust to dural flap and skin sutures to control prolapse at the upper end of the opening.

FIG. 74



The subtemporal, submuscular, decompression operation of Cushing, showing method of separating the temporal muscle and of biting the bone away with forceps. The dura has been opened.

FIG. 75



Subtemporal decompression with bulging hernia.

The chief fault we have to find with Cushing's method is that the area is not always a silent one. Paresis of the face and arm occur at the lower end of the Rolandic area is not rare, and we have seen one case in which the leg centre was involved in the resulting prolapse.

Cushing himself now uses a straight vertical incision in place of the flap, but we still prefer the older method.

(b) **Suboccipital Decompression.**—Suboccipital decompression is a very useful procedure for tumors below the tentorium and must be preferable in these cases to decompression over the hemispheres. It is recommended for this class of tumors, as well as for the supratentorial tumors, by Horsley.

The important feature of Cushing's operation is that the cerebellum is exposed on both sides at once, so that the entire posterior surface lies free to the view; the whole organ can be dislocated in any direction and especially laterally, so that its other surfaces can be explored, as well as the adjacent basal ganglia, nerves, bone, dura and tentorium. The tentorium may also be incised through this opening and the overlying occipital lobes exposed.

Cerebellar tumors and cysts and acoustic and cerebellopontine angle tumors are well seen and attacked by this method.

The operation has the additional advantage that the bone need not be preserved, the thick nuchal fascia and muscles serving as thoroughly adequate protection to the cerebellum against postoperative prolapse and trauma. It is easy to preserve the periosteum here and, eventually, there may develop some new bone, though not often.

Technique.—The tourniquet cannot be applied to this locality, so that other measures for hemostasis must be relied upon, chief among which are rapid operating, pressure on the flaps by an assistant and prompt catching and twisting, or ligating of bleeding points.

Incision for Suboccipital Decompression.—We use the curved incision of Cushing in preference to that of Krause, but find his additional incision in the median line rarely necessary. A horizontal cut is made through the skin and galea at the level of the external occipital protuberance by Cushing, and this incision is continued two and one-half to three inches on one side of the middle line. When the cross-bow incision is employed a cut is made in the middle line through skin, nuchal muscles, and fascia down to the level of the second or third spinous process.

Hemorrhage at this stage is free and is best controlled by rapidly pushing the flaps up from the bone, turning them back, and having an assistant exert pressure on them. The assistant may also catch the chief bleeders with Kocher's forceps while he is exerting pressure on the flaps.

A trephine opening in the occipital plate is then made and the De Vilbiss forceps used to enlarge it, and ultimately the rongeur forceps also, since there is no particular object in preserving the bone in this well-protected locality.

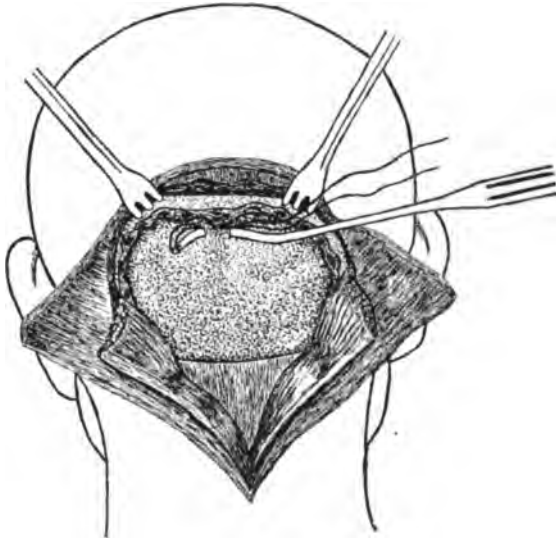
Particular care must be taken to avoid wounding the venous sinuses

or, when it becomes necessary to cut through them, to ligate them in advance.

The occipital sinus is usually impossible to preserve, but the transverse sinuses must be spared, at least on one side. The sinuses are best protected by separating the dura from the bone with a Braatz or similar probe, well in advance of the rongeur forceps.

When the bone has been removed the whole posterior surface of the cerebellum lies exposed to view and may be easily dislocated in any direction to assist exploratory and operative procedures.

FIG. 76



Method of ligating the occipital sinus and the overlying strip of dura, when it is necessary to carry the dural incision across the midline.

Closure is as previously described for any decompressive operation.

Acusticus tumors are best attacked by this method, the dislocation of the cerebellum to one side allowing of a very free exposure of the cerebellopontine angle and permitting of total extirpation of tumors, parts of which might easily have been left behind in the old unilateral procedure, to say nothing of the much diminished danger of trauma to the cerebellum and danger of shock, collapse, and respiratory failure from pressure on the medulla. It is also much less frequently necessary to sacrifice part of the cerebellum in order to enlarge the field, as was the case in the older operations.

(c) **Special Procedures in the Removal of Cerebral Tumors.**—Certain varieties of tumors require special methods of attack.

1. **Cysts.**—Cysts unless they lie in the cortex are usually located by exploratory puncture or exploratory incision of the cortex. After they have been located by the needle mere aspiration is, however, usually not sufficient for a cure. If left to themselves they are very apt to

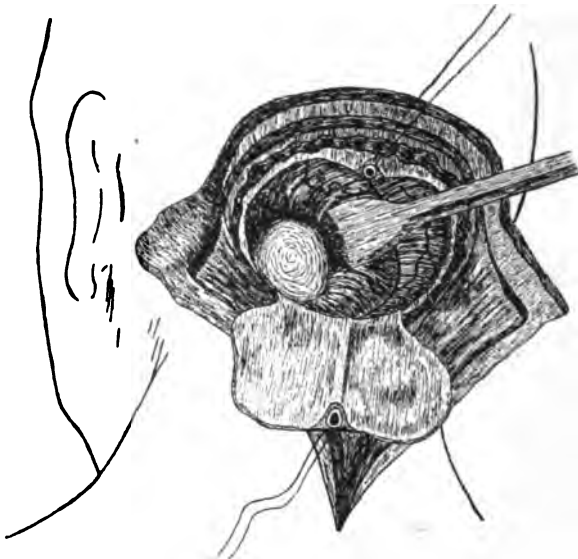
refill and cause a recurrence of the symptoms. Therefore, every cyst should be well opened up and packed or drained, so that it will have an opportunity to become obliterated.

If there be present a well-marked cyst wall, it should, as a rule, be excised, if possible, or at least curetted. Enucleation is imperative in the case of parasitic cysts.

The entire wall of the cyst should be carefully explored before the packing is inserted, so that any tumor nodes will not be overlooked. Certain tumors, *e. g.*, glioma and sarcoma, are very apt to undergo cystic degeneration, which may reach such a high grade that the tumor itself is entirely overlooked, so greatly does the cyst development dominate the picture.

In the case of parasitic cysts the subdural space should be well walled off with iodoform gauze, as in the case of abscess drainage, and the wound and cyst cavity should then be irrigated with some antiseptic solution, *e. g.*, bichloride, 1 to 2000, before the wound is closed. Macewen uses 1 per cent. carbolic acid and also powdered iodoform.

FIG. 77



Serous cyst of the cerebellum, near the cerebellopontine angle. (Author's case. Recovery.)

2. Tuberculoma.—Tuberculoma should be treated, as a rule, as a tumor. When possible, the whole mass should be dissected out into normal tissue and the wound washed out with an antiseptic, *e. g.*, carbolic acid, 0.5 per cent., before closing it.

If this procedure is not practicable, owing to size or location of the tumor, or the condition of the patient, the mass may be scraped out with a sharp spoon, irrigated with a carbolic solution, packed with

iodoform gauze, and allowed to close by granulation. It is, of course, just as necessary to pack off the subdural space in such a case as for abscess or parasitic cysts.

One unfortunate characteristic about tuberculomata is the frequency of their multiple occurrence. It is the rule rather than the exception after the successful removal of a conglomerate tubercle to have the patient die relatively soon afterward, either from generalized tubercles of the brain, or from tuberculosis elsewhere in the body, especially in the lungs. For these reasons some of the operators have gone so far as to say that operation is not indicated in the case of tubercle, even though localizable. This position is illogical, in view of the by no means infrequent cases of long-standing cure; for we know that without operation these patients are surely lost.

3. **Syphiloma.**—Syphiloma, when producing focal symptoms and not yielding promptly to specific treatment, should be attacked like tuberculoma and excised. Horsley irrigates the site of the excision and also the meninges with bichloride of mercury, 1 to 1000. Thus far he has not seen any subsequent toxic effects from its use. Drainage afterward is scarcely necessary, as it is with tuberculoma, but specific treatment should be continued during convalescence and until all the symptoms, including a positive Wassermann reaction, have disappeared. In these cases, both before and after operation, we still continue to give mercury rubs combined with potassium iodide internally, and in addition give a salvarsan injection of five-tenths or six-tenths decigrams, according to the weight and condition of the patient, as soon as the diagnosis has been made positively. The intramuscular injection of a slightly alkaline solution is employed, because it seems safer than the intravenous injection, and thus far has shown no ill-effects. It is to be hoped that Ehrlich's new neutral preparation of salvarsan, the so-called neo-salvarsan, will be still more favorable for this intramuscular method. That it is a *therapia magna sterilisans* we no longer believe and therefore repeat the dosage from time to time when the patient's condition demands it.

Temporary decompression for unlocalizable gummata which fail to respond promptly to specific treatment is sometimes of great value in preserving the sight when amaurosis is threatening in consequence of choked disk or optic neuritis. Cushing first called attention to this fact in 1909 and since that time it has received ample justification at the hands of other clinicians. Horsley's irrigation of the meninges with a mercury salt might also well be tried at the same time. The value of the decompression is probably chiefly in eliminating the pressure on the optic nerves due to secondary hydrocephalus, and it is therefore possible that one of the other puncture procedures for hydrocephalus might be as efficient as the more severe procedure of trephining. In this connection we may mention the observations of Koellner, that scotomata for blue or violet are very frequent in cases of syphilitic neuritis (in 60 per cent.), and also occur very early in the course of the affection. Such scotomata should always be searched for, and when

found, should indicate the use of vigorous antisyphilitic therapy in the absence of contraindications.

Krause's Fixation Method.—In connection with the technical details of tumor removal should be mentioned Krause's method of fixing them by means of suction before attempting to dislodge them. This he does with a hollow glass tube, which he connects to a suction apparatus by means of rubber tubing. The glass tube is applied to the surface of the tumor to be grasped. The tumor is then sucked partly up into the tube and held there by the force of suction while it is being freed from its connection with the surrounding brain tissues. The object of the suction is merely to grasp the tumor for the purpose of assisting in its removal, and not to draw it out of the brain by the force of suction alone, as some writers wrongly have understood. This technique has the advantage over grasping the tumor with fingers or forceps in that it does not macerate the tumor, and produce hemorrhage, and that it greatly aids in drawing the tumor outside the brain cavity so that its base can be more easily reached. Only very soft sarcomata are likely to tear by this method, and they break up with any kind of handling. This technique is applicable to almost any brain tumor, but is especially valuable with those which lie loosely imbedded, such as acusticus and hypophysis tumors, and other deep-lying ones. When used for acusticus tumors or others in the posterior fossa, care must be taken not to make the suction so strong as to draw the pons or medulla into the tube. To avoid this calamity, and in order to permit of prompt cessation of the suction, the tube is provided with a small hole over which the finger is kept as long as suction is desired. As soon as the finger is removed the air rushes in and the suction at once ceases. This method appears to me to represent a distinct advance in the handling of intracranial tumors, especially those of the cerebellopontine angle and hypophysis.

THE OPERATIVE TREATMENT OF EPILEPSY

From the surgical standpoint, epileptics may be divided into two fairly distinct classes: (A) those who present focal brain symptoms, and (B) those who do not.

A. In the first class are included those cases of (1) pure Jacksonian epilepsy in which the recurring attacks of epilepsy always or nearly always begin in the same location in the body, on one side of the face, in the arm or leg, hand or foot, the patient otherwise showing no focal symptoms, and (2) cases of epilepsy of either the Jacksonian or the idiopathic type, which also present focal brain symptoms (symptomatic epilepsy). Both these classes of cases require intracranial operations for their radical relief and offer a fair chance of a favorable result.

B. "Idiopathic" epilepsy. This class of cases is constantly becoming more and more restricted as the development of neurological diagnosis enables us to pick out new organic groups. The cases in

which we are unable to make any pathologico-anatomical diagnosis *intra vitam* still constitute, unfortunately, the major bulk of the cases of epilepsy, and although in some cases operative procedures on the brain have been followed by permanent cures, and somewhat oftener, by longer or shorter remissions, the operative outlook is relatively poor.

Pure Jacksonian Epilepsy.—The first *sine qua non* for operation is the establishment of the Jacksonian character of the epilepsy. The statements of the relatives upon this point should not be relied on entirely; but the diagnosis should be based upon medical observation. For this purpose a hospital stay is usually necessary. In case the attacks are occurring frequently there will be plenty of *chance* for sufficient observation. In case the attacks occur only at rare intervals, there is always plenty of *time* for observation, because there is then no need to hurry the operation.

The Site of the Operation.—Since in the present state of our knowledge we must consider all epilepsy as cortical in origin, we will perform our trepanation, in such cases as show well-marked Jacksonian symptoms, over that part of the Rolandic area which is indicated by the character of the attacks. Other local guides which may be present we shall regard as of secondary importance, such as old irregularities of the skull, history of local trauma, and so forth.

The Operation.—After making the usual Wagner flap over that part of the Rolandic area to which our symptoms point, and raising the dural flap, it remains to explore the surface of the brain for pathological changes. These changes, if found at all, are usually seen lying superficially, such as old scars, thickening of the pia, plaques jaunes, remnants of hemorrhages, cysts, scleroses (usually after encephalitis), tumors, and the like. If such gross changes are found, they should be removed by excision, and the skull closed without drainage, save in the presence of pus or tuberculoma. In case no gross change is found at once, the brain, in addition to being inspected, should also be palpated carefully, and a needle plunged into any suspicious areas. In this way cysts and tumors may be found occasionally, which would otherwise escape observation. The occurrence of abnormally dilated vessels should be looked for as a guide to the location of pathological changes. In case, even under these circumstances, no pathological lesion be found, recourse should be had to faradic localization. The use of unipolar technique, with a galvanic cell and a coil, is recommended, because it permits of more accurate localization. The faradic method is employed because it is safer than the galvanic. It is not necessary to strip the brain of the pia before using the current. The damage done to the cortex by this stripping more than offsets in value the readier reaction to stimulation which is the result of the stripping. The surface of the brain, is dried, if wet, and multiple punctures are made in the arachnoid, if it be edematous. One area after the other is tested, using a very weak current at first and slowly increasing its strength until the reaction is produced. In order not to overlook the local twitching

reactions, it is necessary to have at least three observers: one for the face, one for the arm, and one for the leg. Great care must be taken not to use too strong a current, because of the always present danger of inducing an epileptic attack. When the primary epileptic focus has been discovered it should again be carefully examined, explored with the needle, and if any gross change be found it should then be excised; the vessels leading to it being ligated in the same way as in case of the excision of superficial tumors. As a check on the operation, the excised piece should always be examined, microscopically, by a competent pathologist. Krause has demonstrated that many of these cases show marked gliosis, microscopically, when they show no gross changes. The dura should then be sewed up and the osteoplastic flap replaced. The after-treatment is not different from that after any other intracranial operation.

It is almost needless to add that the prognosis given to the relatives in all these cases should be very guarded. In many cases, and more especially when there is a gross lesion present, which it is possible to remove, there is a temporary improvement and sometimes a complete and permanent cure. Unfortunately, in some cases, especially those in which no gross pathological change is found, the patient's condition after the operation returns to what it was before.

Symptomatic Epilepsy.—What we have said above applies in the main also to the treatment of symptomatic epilepsy. However, in a pure Jacksonian epilepsy we always trephine over that part of the Rolandic area indicated by the character of the attacks, we may trephine here primarily over some other area in case the symptoms point strongly enough toward it. If we trephine elsewhere than over the Rolandic area, the use of faradic stimulation is, as a rule, dispensed with. Its use is purely experimental under these conditions.

Epilepsy following Trauma.—Epilepsy following trauma, especially skull fracture, is often the result of the pressure of a hematoma or a cicatrix following its organization, or of a splinter of bone. Such cases should always have a careful x-ray picture taken directly after the accident, and unless all the symptoms caused by the accident promptly disappear, the exploratory operation should be performed. The evil results of waiting until the hematoma has become organized into a cicatrix and the epilepsy become well established is too well known to require special elaboration.

Genuine Epilepsy.—Under what circumstances is an operative procedure justified in cases of genuine epilepsy? We are forced to admit that the operative prospects in such cases have made no material improvement in recent years. Kocher and Krause do a simple decompression in such cases when they can secure consent, the procedure being based on the observation that many patients with epilepsy have an increased intracranial pressure at the time of the attacks. Their experience has not been gratifying. Krause justifies the operation on the basis that he has seen many "improvements" following it. But we still look askance with von Bergmann at so-called operative "improve-

ments," and prefer to count as successes only those cases which are definitely and permanently relieved. The rareness of such cases is admitted by all operators. In the cases in which there is a diffuse sclerosis of the cortex (Chaslin's gliosis) there is no prospect of any success at all, and we can never tell, certainly, that we have not such a case before us. The presence of dementia, or of a distinctly demonstrable mental deterioration, is therefore particularly suggestive and practically always precludes the possibility of complete operative success.

Reflex Epilepsy.—Since we have come to regard epilepsy as an expression of pathological activity on the part of the cerebral cortex, there has been a marked tendency to drop the conception of a reflex epilepsy of peripheral origin. Excision of scars of the soft parts and peripheral amputations, much done by Dieffenbach and his school, have now passed out of mode. When the epileptic attack begins with an aura located in such a lesion, we might still conceivably do such an operation in the hope of thereby influencing the attacks. Such peripheral sources of irritation as phimosis, hemorrhoids, projecting nasal spurs, hypertrophied tonsils and turbinates, ingrowing toe nails, and the like, should be removed in the epileptic, as in the non-epileptic. In some cases there has been noticed a remission or cessation of the attacks after such minor operations. Krause reports a case of epilepsy combined with trigeminal neuralgia of all three branches, which disappeared after the resection of the Gasserian ganglion. To regard every peripheral scar or even those adherent to bone or nerve as capable of setting loose epileptic attacks is driving the conception of reflex epilepsy to an absurdity. All cases of epilepsy should be subjected to a course of internal treatment before coming to operation. A not too prolonged course is advisable, even in the cases in which the epilepsy is only one symptom of a focal lesion of the brain. By such a preliminary course of treatment the occasional cases of postoperative exacerbation of the epilepsy may be made rarer.

TRAUMATIC LESIONS OF THE SKULL AND BRAIN

Since this article deals primarily with neurological surgery, we do not feel justified in going into the subject of traumatic lesions to any considerable extent. This is chiefly emergency surgery and belongs to the general as well as to the special surgeon. It is well handled in the text-books on general and emergency surgery. To von Bergmann's masterly article in the first volume of his work with von Bruns, and to the article in Lejar's *Emergency Operations*, I will refer the surgeon who is particularly interested in these cases.

We operate immediately in all cases of *fracture of the vault*, or suspected fracture, which show local or generalized symptoms of pressure on the brain. We consider this practice much superior to the expectant treatment, not only as to immediate, but also as to ultimate results.

While the operative treatment of fractures of the vault has long

been on a fairly satisfactory surgical basis, we still remain relatively helpless before those on the base, because of the relative inaccessibility of this region, and the difficulties of accurate localization of the associated lesions of the brain.

The subject of *basal fracture* has of late attracted a good deal of attention. Quenu puts his faith in lumbar puncture. One of his students (Muret) reports 20 cases, in 17 of which the patient recovered in very short order following this treatment. According to Muret, the diagnosis as well as the treatment by spinal puncture leaves almost nothing to be desired in the way of favorable results. E. Vincent, of Algiers, recommends trepanation in all cases. It should be done as near as possible to the base, the dura opened, and the subarachnoidal space drained of blood and liquor, and the drainage continued for twelve to fifteen days. Since these basal fractures practically always communicate with non-sterile cavities, Vincent thinks that he will avoid infection by removing the bacterial pabulum. He reports two cases, one successful and one ending in encephalitis two months after the operation, as the result of "neglect in keeping up adequate drainage." In already infected cases he recommends the trepanation and drainage of the meninges according to Auvray, but admits that there does not seem to be much prospect of success in such cases. Sir Victor Horsley recommends temporal craniectomy to the same end. Cushing recommends early decompression by trepanation. H. Luxembourg reports five cases from Bardenheuer's clinic in Cologne. These were all treated by making a large osteoplastic flap in the parietal region and clearing out the intra- and extradural clots. The results were good. Payr has recently discussed the whole subject of skull fractures at some length. For the basal fractures he recommends Cushing's subtemporal decompression.

Our personal experience has been against intracranial operations in the majority of these cases of *fracture of the base*. The cases with the outcome of which we have been most pleased have been, with few exceptions, those in which we carried out only conservative and palliative treatment, absolute rest, with head high, urotropin freely, plugging the ears with sterile cotton, rectal feeding for the first few days, in the cases where the fracture has perforated into the mouth or pharynx. In the cases with continued bleeding and rapid increase of intracranial pressure the only rational treatment, we believe, is the operation where the skull is opened and the bleeding vessel sought and treated according to well-known surgical principles. It goes without saying that after the arrest of the hemorrhage, blood-clots and crushed brain tissue should be removed and adequate drainage provided. In dealing with intracranial hemorrhages it must not be overlooked that an injured venous sinus may be the source of a hemorrhage that cannot be differentiated clinically from hemorrhage from the middle meningeal. For this reason we do not approve of the procedure of ligation of the carotid or its branches outside of the skull for the treatment of intracranial traumatic hemorrhages.

In three cases in which the diagnosis of middle meningeal hemorrhage was made by a neurologist, we found at the operation, in one, an injury to the jugular bulb; in two others an injury to the lateral sinus just distal to the sigmoid bend. In none of these did there exist any concomitant lesion of the meningeal, although a history of "a lucid interval" was distinct and formed a part of the classical picture of a ruptured middle meningeal artery.

Another reason for advocating cranial exploration in all cases of suspected hemorrhage is that in extracranial operations we cannot remove the clots, nor can we form any estimate as to the amount of damage done to the brain. In fully 50 per cent. of the cases of middle meningeal hemorrhage operated upon, the hemorrhage has ceased when the surgeon operates. The operation then has, for its main purpose, the relief of pressure by removal of the clots and secondarily to provide against renewed hemorrhage. In these cases ligation of the carotid only increases the danger to the patient. In this connection it may be mentioned that brain symptoms due to anemia follow in 25 per cent. of all ligations of the carotid, and that death directly results in 10 per cent.

TECHNIQUE OF OPERATIONS ON THE HYPOPHYSIS CEREBRI

The operative technique dealing with tumors of the hypophysis has been a matter of gradual development. As late as 1905, Cushing was led to say that in the near future operations on the hypophysis cerebri might be carried to a successful issue.

A brief review of the steps that have led to our present technique may be of interest in tracing the progress of this most recent triumph of modern surgery.

Methods of Approach.—Of the operations proposed, two principal routes have been chosen to reach the hypophysis: (1) the intracranial; (2) the extracranial or transsphenoidal. Through the former the hypophysis may be reached either by way of the anterior or the middle cranial fossa. The major part of the operation may be accomplished by working outside of the dura, or the dura may be opened in the beginning and the work done entirely within its cavity.

Through the Middle Cranial Fossa.—The possibility of reaching the hypophysis through the middle cranial fossa was first proposed by Caton and Paul, but was not carried out by them. Horsley is generally quoted as following this route.

Stumme quotes a personal communication from Petrien, in which he credits Dahlgren with an operation conducted through the middle fossa. In this the dura was opened and the hypophysis reached by retracting the brain. Nothing is said of the details of the operation or of the result.

Krause suggested what appears to be the most rational intracranial method of reaching the hypophysis. He proposed to open the cranial

cavity by an osteoplastic resection of the frontal bone and to proceed extradurally, through the anterior cranial fossa until the lesser wing of the sphenoid is reached, and then to open the dura and remove the tumor by means of a small, hook-shaped knife devised for that purpose. Krause, from his experience, gained only by operation on the cadaver, believed that by following this extradural route all or most of the obstacles which other operators had encountered would be eliminated.

Borchard attempted to remove a tumor of the hypophysis by the Krause method, and was forced to abandon it because of the hemorrhage. Later, he operated successfully on the same patient by the transsphenoidal route, as practiced by Schloffer.

Killiani, in discussing tumors of the chiasm, suggests an operation which is similar to that of Krause, excepting that the dura is opened immediately after entering the cranial cavity. The longitudinal sinus is ligated, the frontal lobe lifted from the base of the skull, and the tumor attacked above the chiasm and the transverse sinus by means of a sharp spoon.

Temporal Route.—Silbermark suggests a temporal intracranial method similar to that employed by Paulesco and Cushing in their experimental work on dogs. This consists in a bilateral craniectomy, the counteropening being made to permit of the temporal lobe being dislocated without the risk of serious compression. The operation proposed by Silbermark, and by him performed on the cadaver, has never been tried on a living subject.

The relative merit of the intracranial method, as compared to the transsphenoidal, is still a matter for discussion. Most operators have chosen the latter.

The temporal route, by way of the middle fossa, has been followed by Dahlgren and Horsley.

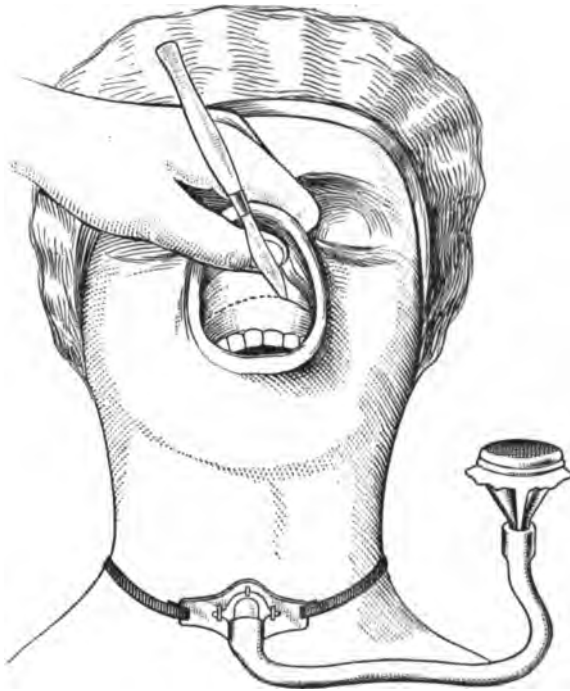
The anatomical structures that come in relation to the operative field are many and of great importance. Injury to the large venous sinuses, if not a serious menace to the life of the patient, would at least delay the operation and would probably preclude the possibility of completing the operation in one sitting. In the intracranial operation, aside from the danger of injury to the important bloodvessels and nerves, retraction of the brain sufficient to expose the hypophysis is likely to be followed by softening or necrosis, as a result of the trauma and interference with its blood supply.

The lateral intracranial operation has been employed by Cushing in over one hundred experiments on dogs. While by this method the canine hypophysis is readily exposed, Cushing is of the opinion that it is not practicable in operations on the human, excepting possibly in benign cysts of the gland that project into the infundibular region. In cases in which the growth occupies the sella turcica, it would be impossible to reach it excepting by the transsphenoidal route.

Transsphenoidal Extracranial Route.—The first to suggest the transsphenoidal extracranial operation was Koenig in 1898. His procedure was identical with the oronasal operation practised by Friedman

and Mass on animals, and by Dussenbauer for the removal of nasopharyngeal growths. It consists in separating the mucoperiosteal covering from a palatal process of the superior maxilla. The latter is then split and the lateral halves retracted. The septum and vomer are removed with the other structures of the nose that may obscure the view of the operator. The sphenoid cells are opened and their posterior walls chiselled away, bringing into view the hypophysis.

FIG. 78



Removal of hypophysis tumor by way of the oronasal route. The upper lip retracted, showing the initial incision through the mucous membrane.

A similar operation has been proposed by Loewe. The nose is split by means of a long incision to one side of the median line, the maxillæ are separated, the inner walls of the maxillary sinus are removed, from the canine fossa upward, and the septum divided back to the vomer, which is removed and the sinuses entered by chiselling away their anterior walls. The sella is reached through the sphenoid cells.

It will be seen that the buconasal route of Koenig and the operation of Loewe are in reality quite similar in the essential features to the later inferior nasal operations. The strongest objections urged against both of these procedures is that they are unnecessarily severe and disfiguring. They have, however, the advantage of offering the most direct route to the sphenoid cells, with least risk of entering the cranial cavity anterior to the sella.

Superior Nasal Route.—The first to suggest the superior nasal route was Giordano. This method, as practised by him on the cadaver, consists in osteoplastic resection of the anterior wall of the frontal sinuses and the nose. These structures are turned down, the ethmoid cells opened and their walls removed. The cells of the sphenoid are then opened and the hypophysis reached through an opening in the anterior wall of the sella.

The operation of Giordano has been followed, though slightly modified at times, by all who have chosen the superior nasal route in operating on the hypophysis.

Schloffer, in 1907, successfully removed a tumor of the hypophysis by a similar method. An incision was made above and to the left of the nose; the whole nose turned down and to the right; excision of all of the turbinate bodies, septum, inner wall of the left maxillary sinus, and part of the left nasal process of the superior maxilla; opening of the ethmoidal cells through which the sella is broken down. The tumor was recognized as a pulsating mass, projecting through the opening in the sphenoid, and was curetted with a sharp spoon. The patient survived two and one-half months. Autopsy showed that only about one-sixth of the growth remained.

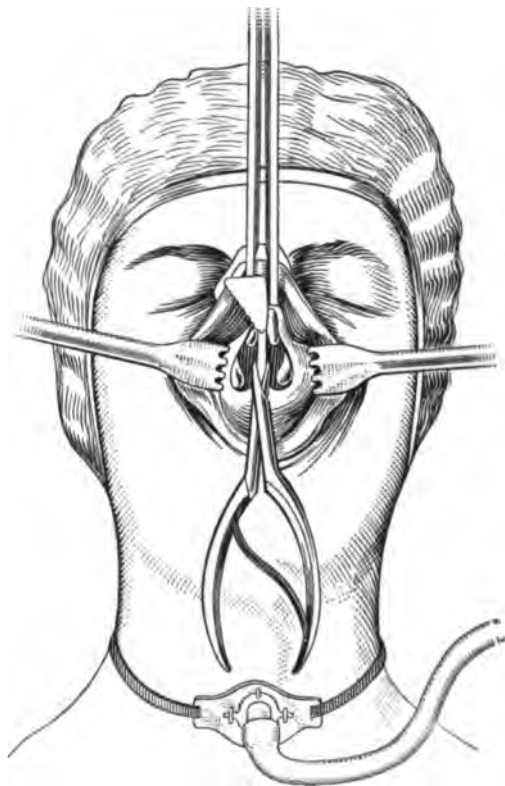
The operation proposed by Moskowicz and Tandler differs slightly from that practised by Schloffer. The inner wall of the orbit and that of the antrum are not disturbed. They also recommend that the operation be performed in two steps. The first takes the operator to the posterior wall of the sphenoid sinus, which is permitted to remain intact. A pedunculated flap of skin is then turned down from the forehead, so that the end comes to lie on the sinus wall. At the second step the posterior wall of the sinus is broken down, and the end of the skin flap pressed into the cavity remaining after opening the sella and removing the tumor. This is held in place by a tampon. The skin flap is so placed, in order to lessen the dangers of infection. This step seems to be of questionable value. It would appear to enhance rather than minimize the risk of infection of the meninges.

v. Eiselsberg, in the three cases originally published, followed the superior nasal route. The incision described differs slightly from that employed by Schloffer, in that a vertical incision along the median line of the forehead meets a curved incision above the root of the nose. The nose, with the anterior wall of the frontal sinus, is reflected downward and to the right. The septum is divided and the superior turbinals removed. The vomer is removed piece by piece up to its root. After lifting the periosteum from the anterior wall of the sphenoid the sinuses are opened by chiselling into them. The tumor is then exposed by removing their posterior walls.

More recently Eiselsberg has reported eleven more cases, making fourteen in all, with four deaths. In his later cases he has modified his technique only by omitting the incision into the frontal sinus. He still turns the nose to one side, and still uses the upper route of approach to the sella.

Stumme, in 1908, describes an operation performed by Hochenegg upon a patient suffering from acromegaly, with other symptoms of pituitary tumor. The technique followed was similar to that of v. Eiselsberg. The anesthetic employed was Billroth's mixture, preliminary to the administration of which the postnares were tamponed with gauze. After turning down the nose, an osteoplastic flap containing the anterior walls of the frontal sinuses was reflected upward. The

FIG. 79



Removal of hypophysis tumor by the oronasal route. Second stage. The septum is divided under the retracted lip and through the mouth. The turbinals and vomer present through the nasal cavity.

septum and the entire contents of the nose, including all of the turbinals, were removed. The inner wall of the antrum and orbit was left intact. The sphenoidal sinuses were opened by means of a chisel and, later, their posterior walls, exposing the tumor, which was removed by a spoon-shaped instrument. The cavity was packed with gauze and the nose replaced and sutured.

In the first case operated on by Cushing, an acromegalic, he employed the supranasal operation, similar in detail to that of

Hochenegg, excepting that the anterior walls of the frontal sinus were turned down with the nose. Preliminary tracheotomy was performed; the nares plugged with a sea sponge. The Rose position was maintained during the operation, as in Hochenegg's case. After rongeur-ing away the walls of the ethmoid cells a narrow mesial channel (two centimeters wide) was chiselled below the ethmoid roof and carried back to the sphenoid cells. The anterior walls of the cells were then broken down and the median projection of the sella into the cells was located. The thin, bony wall of the sella was then clipped away, exposing a pocket of dura enclosing the gland. This was incised longitudinally, and half of the exposed gland removed.

Intranasal Route.—In November, 1909, Kanavel, of Chicago, published a method of approach in removing tumors of the hypophysis that he termed the intranasal route. At the time of publication of the paper the operation had not been performed upon the living subject by Kanavel. After a series of experiments on the cadaver and a trial of the various operative procedures recommended by others, he came to the conclusion that the hypophysis could be best reached by turning up the nose and proceeding directly to the sphenoid cells, without interfering with the ethmoid, after removing the turbinals, deflecting the septum, and cutting away the vomer.

Recently, West, of Baltimore, recommends practically the same procedure, excepting that the operation be conducted through the nose. West recommends that the operation be performed in two stages, and that the preliminary operation of removal of the middle and inferior turbinals be done under local anesthesia. The septum and vomer are removed at the second operation, after which the cells are opened and the sella chiselled through.

Hirsch, of Vienna, has recently published a series of twelve cases of decompression of the sella or partial hypophysectomy, with only two deaths. He does an operation much like West's, but does the resection of the septum and the vomer submucously as far as possible, and aims to use only local anesthesia in the procedure. He also claims priority over West for his operation. When impossible to finish in one stage he uses two or more separate sittings. This procedure may be all right for the stolid Viennese, but we doubt the ability of the average American patient, particularly women, to pass through the long-drawn-out ordeal. Our patients sometimes quail at even the preliminary double turbinectomy when done under local anesthesia by our rhinologist. Hirsch's results are a distinct victory for the lower route. They show the decreased danger of meningitis in this mode of attack, which is very likely due to the lesser frequency of fracture of the ethmoids in cutting out the septum. Through the ethmoid canals run numerous lymphatic channels which connect with the meningeal space. The opening up of these channels to infection is probably the principal source of meningitis. This seems the most logical explanation for the superior results which have been obtained by the lower route when followed either through the nose or through the mouth as compared

with the results obtained from the superior route operations of Schloffer and Eiselsberg.

McArthur has outlined a technique similar to Krause's whereby the hypophysis is approached through the anterior cranial fossa after an incision has been made over the eyebrow. He reports three cases operated by this method. The upper wall of the orbit is removed and the eyeball displaced downward to give a wider approach.

Classes of Tumors of the Hypophysis.—Probably no single line of approach is adequate in all cases. The different operators have so far always clung each to his own method of attack. The x-ray picture now enables us to differentiate three classes of tumors of the hypophysis: First, those growing downward into the sella, and forward toward the sphenoid sinus. This is the type *par excellence* for removal by way of the nose. The x-ray in tumors of this type shows the sella deepened and enlarged but the outlet of approximately normal size. The sella is often seen to bulge forward toward the sphenoid sinus, or even into it. Second, tumors growing chiefly upward and projecting above the sella and optic nerves. In this location it is apt to press upward on the third ventricle, and may thereby cause a secondary hydrocephalus. In the x-ray of such cases the entrance to the sella is seen to be dilated. The dorsum sellæ is often thinned, and it may appear pushed backward, owing to the pressure atrophy produced in the anterior lamella. In the larger tumors of this type we not infrequently see signs of increased intracranial pressure in forms of intracranial erosions or juga cerebralia. These characteristic x-ray findings may be seen sometimes over the entire inner surface of the skull.

Such large tumors, when they extend upward, cannot be removed satisfactorily through the nose. But little more can be done by the intracranial route than to curette away part of their base. The only hope for their radical removal lies in an attack by some such route as Krause's or McArthur's. A feasible method of attack which would give more room in some cases than Krause's route is one which may be pursued in the not infrequent cases in which the roof of the orbit contains a shallow air sinus. The cranial side of the sinus may be chipped away subdurally through an opening like Krause's. The whole frontal lobe is then pushed up and the tumor is attacked from above the chiasm. The dura is not cut until the tumor is reached.

Third, tumors growing chiefly to one side of the sella. In consequence of the unilateral growth one optic nerve suffers much more than the other. The cavernous sinus, and to a less extent the carotid, are liable to be compressed by the tumor, on its side of principal growth. In the x-ray picture in such cases the sella is often shown in three contours. This is particularly marked usually on the anterior surface of the sella, owing to the fact that one side of it is much more eroded than the other side, or even than the middle. Also, the blocking of the cavernous sinus by the tumor leads to the formation of a collateral circulation. This collateral circulation is usually most clearly seen in the dilatation of the sinus sphenoparietalis. The enlargement of this sinus is known

because of the enlargement of its canal in the inner table, the consequence of the enlargement of the sinus itself. This shadow may be seen in the side picture running upward from just above the sella and ending above in the middle line often in a Pacchionian granulation.

A radical removal of such a tumor by the nasal route is a matter of difficulty. Horsley's lateral method of attack seems to us the logical operation for tumors of this sort.

Besides these three fairly well-marked types there are all possible combinations and mixed types. While the intranasal method seems to us the proper route of attack in the great majority of cases of hypophysis, tumor, yet we should always make a careful preliminary study of the x-ray plates and assure ourselves, before operation, of the probable size and direction of growth of the tumor and consequently the most successful route to be followed in a given case. At the same time we can also make some estimate of the probable difficulty of the operation. That type of tumor which shows a greatly dilated sella turcica and presents distinct signs of increased intracranial pressure is usually difficult to successfully remove. It is also the one most likely to be accompanied by a hydrocephalus and dilatation of the third ventricle. A dilated third ventricle, even without any enlargement of the hypophysis, may produce a dilated sella turcica, and does not, at the same time, always show erosions of the inner table. Such a case may be operated under the diagnosis of tumor of the hypophysis. Hirsch has had such a fatal case, the entire intraventricular fluid escaping as soon as his knife penetrated the dilated third ventricle. We cannot recommend too highly to every operator on the hypophysis the careful study of the x-ray plates before the operation.

Halstead's Methods.—The method that I have adopted and carried out successfully in a patient operated upon in July, 1909, and recorded in the *Trans. Amer. Surg. Assoc.*, 1910, is as follows:

Pads of sterile cotton moistened in boric acid solution are held over both closed eyes while the face and neck are washed with soap and water on fluffed gauze and then with alcohol and bichloride, 1 to 1000.

The patient is anesthetized with ether by the usual drop method, the anesthetist having previously scrubbed up, put on sterile gloves, and using a sterile mask. As soon as the patient is asleep, a high tracheotomy is done and a Trendelenburg ballon cannula inserted. Chloroform is now used to continue the anesthesia.

Aditus ad laryngem, pharynx and mouth are now tightly packed with gauze strips.

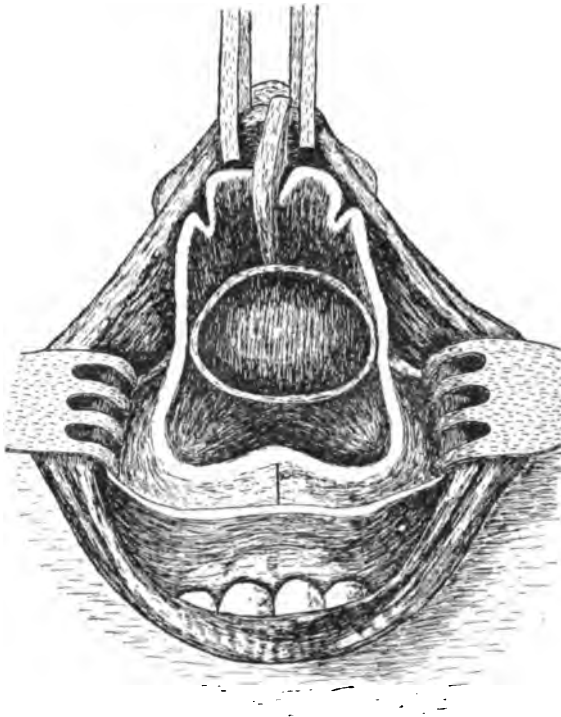
The upper lip is raised and an incision is made in its mucous membrane about five-sixths of an inch from the mucocutaneous junction and parallel to the alveolar process. The soft tissues are freed and the nose gradually drawn up with retractors, the nasal mucous membrane incised, and the nasal cavity opened.

The cartilaginous nasal septum is divided with bone-cutting forceps, flush with the nasal floor and displaced upward and laterally. The lower and middle turbinals, the vomer and the perpendicular plate of the

ethmoid are removed with scissors and forceps. We thus have formed a cylindric cavity of the width of the nose and about four inches deep, reaching the anterior wall of the sphenoid sinus. This cavity is then packed tightly with adrenalin gauze for a few minutes until oozing has stopped and the mucous membrane of the nose and anterior surface of the sphenoid sinuses are well blanched.

Upon removing this tampon the anterior and then the posterior wall of the sphenoid sinus are bitten through with the Hajek punch and the sella turcica with the enclosed hypophysis lies exposed. The tumor is now removed piecemeal with a dull curette, forceps, and scissors. Any subsequent hemorrhage is controlled by tamponing with adrenalin gauze.

FIG. 80



Removal of hypophysis tumor by way of the oronasal route. Third stage. The nose and upper lip are retracted upward over the forehead after dividing the septum, and freeing the soft parts from the superior maxilla. Turbinates, vomer, and all obstructing tissues removed down to the anterior sphenoid cells.

When the tumor tissue has been removed and the hemorrhage controlled the whole cavity is flushed out with normal salt solution and the sella and entire nasal cavity are packed with iodoform gauze saturated with tincture of benzoin.

The septum is then sutured back and the mucous membrane of the mouth reunited by suture and the Trendelenburg cannula replaced with

a tracheotomy tube. The patient is then put to bed in a semirecumbent position. As soon as he regains consciousness the packing is removed from throat and mouth. The tracheotomy tube is removed on the following day and the wound closed by suture and dressing.

The nasal packing is allowed to remain in place two or three days, unless the patient's temperature rises above 100° F. Then it is removed and the nose flushed out daily thereafter with normal saline solution before the packing is renewed. After a week the packing is dispensed with and saline irrigation continued as long as the patient remains in the hospital.

The accompanying figures give a very fair idea of the different steps in the operation and the photograph shows the faultless cosmetic result obtained, which is all the more striking when compared with the results obtained by all the previous methods in which the external incision is used.

The Palliative Trepanation and Exploratory Puncture of the Sella Turcica.—A. Schueller recommends the decompressive trepanation of the sella turcica and also puncture of the third ventricle through the trepanation opening, not only for hypophysis tumors, but also for tumors and cysts of the third ventricle and of the vicinity of the hypophysis and third ventricle. Schueller recommends the nasal approach to the sella, but in performing a puncture of the third ventricle by this route, which is the most important part of his proposal, he suggests passing a more or less flexible probe under the mucosa of the septum, and along it, pushing it through the walls of the sphenoid sinus into the sella, and up into the third ventricle, thus accomplishing from below much the same result that Anton and von Bramann do from above, both procedures being rather blind, but Schueller's has the advantage of draining from the lowest part of the ventricular system, and into a cavity which is always open. These two very distinct advantages may perhaps outweigh the slightly greater risk of infection of Schueller's procedure. The technique seems not more difficult and the other dangers not greater than the Balkenstich. Schueller thinks the puncture will be valuable in chronic external or internal hydrocephalus, whether primarily or secondarily due to unlocalized brain tumors. It ought also to be useful in the lesser grades of hydrocephalus which accompany tower skull and the other forms of craniostenosis and some cases of migraine and epilepsy. This is the only method which can successfully attack localized hydrocephalus or cysts of the third ventricle, or localized meningitis serosa about the chiasm. The operation of decompression is indicated when the x-ray shows a deepening of the sella along with the other signs of increased intracranial pressure, and a sufficiently roomy sphenoid sinus to allow of free decompression. If only a simple puncture is to be done, the size of the sphenoid sinus, of course, cuts no special figure. The simple decompression is not accompanied by the same danger of infection as the removal of the hypophysis, because in the former procedure the dura is left intact.

Anton has criticised the sellar puncture of the third ventricle as being

PLATE L



A Patient (H. R.) Two Months after Removal of a Tumor of the Hypophysis through the Oronasal Route.

1901

too dangerous, on account of its relation with the nose. Anton apparently is not familiar with or has not read Schueller's original article, because Schueller by his technique does not *enter* the nasal cavity—the procedure is entirely submucous when typically done and abnormal anatomical relations do not greatly interfere.

Canestri and von Saar have also suffered from a misapprehension of Schueller's idea, in that they have tried to do the simple sellar trepanation by the old mutilating operation of Schloffer, instead of by the newer and simpler technique of Hirsch or Halstead. They therefore had a failure. There have been no cases operated yet by the strict Schueller technique. Until there are it will be premature to form a final opinion regarding it.

Chordoma.—Before leaving the subject of hypophysis tumors, it may be of interest to speak of chordomas in this connection. These are vestigial tumors, springing from the upper end of the embryonic notochord. They are most frequently seen, as an accidental finding, at autopsy, usually a small whitish excrescence in the bone of the clivus Blumenbachii, or perhaps perforating it, and appearing in the middle line in the posterior fossa. These are the benign chordomas. In some cases, however, these rare tumors may become malignant and then they may perforate into the sella turcica, and even through the base of the skull into the upper pharynx. The benign tumors have never as yet been diagnosticated clinically, but two of the malignant variety have been discovered after operation, and one of them perforated into the pharynx.

The case of Spies was a cyst which was operated under the diagnosis of hypophysis tumor; the diagnosis of chordoma was made from a microscopic examination of fragments of the cyst wall.

The case of Stenger was operated under the diagnosis of malignant tumor of the nasopharynx. The character of the tumor was entirely unsuspected until the microscopic examination was made. The patient died two years after the operation from recurrence, and no postmortem was obtained. But at two subsequent operations for the recurrence, the tumor tissue removed, showed each time the characteristic structure of chordoma.

These two cases are, so far as we have been able to determine, the only ones of chordoma which have ever been attacked surgically. But since the hypophysis is now being frequently operated, we shall probably hear soon of more of these cases, and every operator should be on the lookout for them. Hypophysis tumor, the x-ray of which shows complete destruction of, or even marked involvement of, the clivus Blumenbachii, should be under suspicion.

Tumors of the Pineal Gland.—Following the discussion of hypophysis tumors it seems proper to mention the rather rare tumors of the pineal gland, which in some ways are the counterpart of the tumors of the hypophysis. That certain tumors of the pineal gland, especially teratomata, are sometimes accompanied by such unusual symptoms as increased body height, abnormal growth of hair, premature genital

and sexual development, has been known for a long time, and such cases have been reported by Gutschik, Oestreich and Slawyk, Jonkowsky, H. Nothnagel, and E. Mueller. Otto Marburg, of Vienna, however, deserves the credit of first placing the pineal gland among the glands of internal secretion, and, on the basis of its functions, he has separated its disturbances into three distinct clinical groups, using for that classification one case of his own and others from the literature. His divisions are: (1) *Hypopinealism*, characterized by abnormal increase in height, premature development of the primary and secondary sexual characteristics, and precocious sexual ripeness. (2) *Hyperpinealism*, the chief symptom of which is adiposity. (3) *Apinealism*, the symptoms of which are rather indifferent, save that a marked cachexia is often seen.

Of all these pineal symptoms the most striking are the early sexual ripeness and the precocious development of the primary and secondary sexual characteristics. On the basis of these symptoms in connection with other symptoms of brain tumor, Fraenki-Hochwart was able to make the diagnosis of pineal tumor *intra vitam*. Still more recently Bailey and Jelliffe, of New York, have gone over the entire subject with great thoroughness, and have been able to collect fifty-nine cases from the literature, including one of their own, a boy of twelve years, with a teratoma.

As an additional fact of importance it should be mentioned that the pineal gland may often be seen in the x-ray picture, especially in the side view, owing to the fact that calcium salts are early deposited in it, and sometimes in considerable quantity. The patients with such calcified epiphyses are, however, usually on the far side of puberty, while the patients with tumor are usually in their early years.

Not only is the diagnosis of pineal tumors now on a fairly rational basis, but its surgical removal or destruction has been done in animals, though not yet in man. The operation even on the animal is one of great danger to the subject. Exner was able to save only 22 out of 95 rabbits operated upon by him.

Biedl and Sarteschi had no better success. The results of the extirpation in the animals who survived the operation were, however, for the most part negative. No apparent change at all in the nutrition, weight, sexual life or sexual characteristics could be noted by Exner.

No one has yet suggested a mode of attack for such a tumor in man. A procedure similar to Anton's Balkenstich may be attempted, perhaps with the assistance of an endoscope. By entering in the middle line just behind the motor area the falx could be followed downward and then by perforating the corpus callosum one might come directly upon the tumor. This could then be destroyed by the electric cautery under the guidance of the eye, or might be sucked up and then removed by Krause's method. This method would also have the advantage of enabling the operator to open a ventricle at the same time, and provide for escape of the ventricular fluid, in case the tumor had blocked the fourth ventricle and caused hydrocephalus.

**DANGERS AND COMPLICATIONS OF INTRACRANIAL OPERATIONS,
THEIR AVOIDANCE AND TREATMENT**

In general, these dangers and complications are not many and are relatively easily avoided, as a rule, if borne in mind during operative proceedings. Most of them have been already touched upon during the previous general discussion of the subject of operations.

Dangers at the Time of Operation.—1. **Hemorrhage.**—Hemorrhage has already been discussed at sufficient length under operative technique. It may at times be annoying, but never dangerous, with our modern means of hemostasis—tourniquet, Kocher forceps, preliminary sutures, pressure, tamponade, Horsley's wax, muscle tissue, orange sticks, Jap toothpicks, etc.

2. **Shock, Collapse, and Sudden Respiratory Failure.**—These complications are now much less frequent since the introduction of the principle of cerebral dislocation and the two-step operation, and the recognition of the fact that these alarming complications are due to sudden changes of pressure, chiefly overpressure on the medullary centres.

Though prevention is the main factor in their treatment, we consider of great importance the intravenous administration of normal salt solution, with the addition of 2 c.c. of 1 to 1000 adrenalin solution to the liter in amounts up to two to four liters. Hypodermoclysis is also of value, but is too slow for the cases of sudden collapse. The use of the reversed Trendelenburg position is also important. For the operation itself the head and shoulders are kept high as long as the patient's condition is good, but as soon as collapse threatens they should be promptly lowered.

We have at times considered the advisability of laying bare the internal saphenous vein before starting the cranial operation in cases in which there is reason to anticipate collapse, so that salt solution might be given in large amounts and without a moment's delay.

3. **Infection.**—Septic infection is practically never introduced from without in the modern, aseptic operation. It can occur only when we have to deal with tissues already infected, *e. g.*, subdural abscess, cerebral abscess, and purulent meningitis, the last being rarely attacked by operative procedure, or occurring with the wound of the cranial vault.

The avoidance of infection in the treatment of intracranial abscess has been discussed already under the subject of brain abscess. It consists in (a) puncturing the brain to locate the abscess only after the dura has been opened and the subdural space walled off with several layers of iodoform gauze; (b) thorough opening of the abscess; (c) irrigation followed by renewal of the outer layers of gauze, and (d) drainage puncture through the galea flap directly over the abscess opening.

4. **Danger from Parasitic Cysts and Malignant Tumors.**—Danger of infection of the meninges and brain tissue from parasitic cysts and malign-

nant tumors or granulomata is to be combated in the same way as in the case of intracranial abscess, except that in the former case removal of the source of infection by excision into healthy tissue may at times enable us to get rid of the entire focus of infection at one stroke.

5. **Dangers from Anesthesia.**—It goes without saying that in this field of surgery, if in any, the assistance of an experienced anesthetist is required. The awkward, strained position which the anesthetist must assume in operations on the posterior half of the cranium, enshrouded in a sheet and breathing vitiated air saturated with ether, with irrigating fluids trickling down on him at intervals, makes his duties onerous. He must administer the anesthetic to a point above his head, keep irrigating fluids out of the patient's mouth, nose, and eyes, hold up the mask continuously and occasionally, when another assistant is not available, watch respiration and heart beats through a Bowles' stethoscope fastened to the patient. In these exacting cases I feel that such good results as I have obtained have been due in no small measure to the experience and skill of my anesthetists at St. Luke's Hospital.

Shock and collapse are to be avoided by care, not only on the operator's part, but just as much by careful watchfulness, backed by experience on the part of the anesthetist.

To prevent vomiting, washing out of the stomach, just before the operation and just after the patient has regained consciousness is the most efficacious measure, though not any too agreeable to the patient.

Post-operative Dangers and Complications.—1. **Collapse.**—Collapse, though not prone to occur in the late stages of the operative procedures, may appear at any time, even several days after the operation; but the danger is greatest in the first twenty-four to thirty-six hours after the operation.

The remedial measures are the same as when it occurs during the operation, except that, inasmuch as the collapse does not appear in as urgent a fashion as in the latter case, it allows the surgeon more leeway in which to act.

2. **Postoperative Hemorrhage.**—If in addition to collapse there are pressure symptoms, there is probably present a postoperative hemorrhage which, if extensive or progressive, must be treated by reopening the wound, scraping and irrigating away the clotted blood, and ligating or tamponing the bleeding point.

Hemorrhage is best prevented. Careful hemostasis during the operation, ligation of all bleeding points and tamponing for a day or two, of any oozing beyond the slightest amount, will save the surgeon worry and his patient a second operation.

3. **Edema and Softening of the Brain.**—Edema and softening of the brain (encephalomalacia) may be the consequence of a neglected hemorrhage, or it may arise after trauma of the brain substance. It varies, as a rule, directly with the amount of injury to which the brain tissue has been subjected during the operation, and is commonly most severe after removal of subcortical and extensive tumors and cysts, in the

treatment of which it is often necessary to interrupt the continuity of many nerve fibers and bloodvessels in brain tissue whose vitality has already been lowered by the long-continued presence of the tumor.

The most severe grades of edema which may cause prolapsus or even fungus cerebri and extensive paresis and paralysis are fortunately rare, for they may be rapidly fatal.

Slight edema associated with a superficial necrosis of the brain tissue surrounding the tumor, and a small discharge of broken-down brain tissue on the third or fourth day following operation, without other symptoms of moment, is less infrequent and has no particular significance.

4. **Prolapsus and Fungus Cerebri.**—The former complication is common and is to be expected whenever a decompression operation is done for increased intracranial tension at a spot where the galea is not reënfored by muscle, *i. e.*, outside the subtemporal and suboccipital regions. It is at that result that we aim. It has, as a rule, no untoward symptoms, unless excessive, and can, until it reaches a considerable amount, be concealed by hair-dressing, particularly in women.

Fungus cerebri may arise when (a) intracranial pressure, already excessive at the time of operation, is still further increased by post-operative edema; or when (b) a decompressive operation is done directly over a rapidly growing tumor, especially in an area not covered by muscle; or when (c) tamponade must be continued for several days, on account of hemorrhage or suppuration, and when intracranial pressure at the same time is excessive.

The treatment is, first and foremost, prevention. Tamponade, as a hemostatic measure, must be avoided as far as possible in cases of increased tension. When tamponing is unavoidable, the protecting bandage over the skin flap should be applied tightly, to reënfice the skin approximation. The packing should be removed and skin stitches inserted at the earliest advisable moment, within thirty-six to forty-eight hours if possible.

Abscess drainage should be led directly through a punctured wound in the skin flap, so that the suture is left undisturbed. This drain should be tubular, not bulky, therefore allowing much less chance for subsequent prolapse than does tamponade. The tamponade of the subdural space in abscess is the chief source of danger from prolapse in such cases, since it can only be removed gradually, when we feel certain that adhesions have formed that protect the space.

Decompressive trepanation should not be done directly over a tumor, particularly a rapidly growing one. At times this procedure is unavoidable, because we cut down on the tumor site, hoping to be able to remove the tumor, and, finding it impossible to eradicate, we are forced to turn the operation into a decompression in order to relieve the symptoms. But if we have an osteoplastic flap of such shape that it may be returned accurately in one piece to the cranial defect, and if the patient's condition is sufficiently good to allow of a second decompressive trephining at one of the sites of election, *i. e.*, subtemporal or suboccipital

(a combination of circumstances which too frequently does not obtain), we may observe the rule at the expense of prolonging the operation and increasing shock. In such cases a point in technique of considerable value is to make the dural opening at the base of the galea flap, so that there may be a certain amount of overlapping, which will keep the prolapse well covered when it does occur, and also to a certain extent protect the skin suture line while it is healing.

The exertion of pressure over the skin flap is in general to be carefully avoided, but becomes a necessity when prolapse is considerable and increasing and threatening to result in a fungus.

When a fungus has actually formed (a) it must be kept scrupulously clean, fresh dressing being applied daily with careful asepsis; (b) pressure must be applied by the dressing in an attempt to reduce the swelling or at least to keep it within bounds; (c) tension sutures (silk-worm gut) may be applied to draw the skin flaps together; or (d) an attempt may be made at a plastic closure by using a galea flap, but the success of this latter procedure is always doubtful in rapidly developing fungus. Lastly, (e) excision of the fungus may be practised to keep it within bounds or from becoming infected. It is remarkable what large areas of the "silent regions" of the brain may be removed in this way without producing any appreciable symptom. In cases of doubt as to whether to excise or not to excise, it is well to remember that it is much better to cut away such a portion than to have it slough away, with the consequent dangers of local and meningeal infection.

5. Escape of Cerebrospinal Fluid.—Escape of cerebrospinal fluid in considerable amount through the wound is not at all unusual. If it does not occur through the wound itself, it is very apt to trickle through a needle puncture somewhere along the line of suture, and, once started, it may be very persistent. For this reason I make it a practice to remove the stitches as early as possible in my cranial operations, usually from the fifth to the seventh day, if the wound is in good condition, and apply a snug dressing for a few days after the stitches have been removed, and usually, also, collodion and tincture of benzoin on gauze to the suture line.

As long as liquor trickles anywhere through the wound, careful aseptic daily dressing must be continued, and during the day the outer dressings must be renewed by the nurse or interne as often as they are saturated with the discharge.

The sealing of the line of suture with collodion at the operation and the subsequent daily dressing with tincture of benzoin or collodion on gauze strip is, I believe, of considerable assistance in preventing these small leaks.

When a leak has already occurred, it is not much use to try to put a stitch through it. New portals of exit are too apt to form through the needle punctures.

I usually cauterize these small openings daily with the silver nitrate stick and then apply gauze strips saturated with tincture of benzoin, with compression by the dressing or adhesive strips over that. Prac-

tically all these small fistulas yield to this treatment in the course of a week or two.

Occasionally the closure of one of the more obstinate and persistent of these little fistulas may be accompanied by renewed symptoms of intracranial pressure of low degree, headache, malaise, nausea, etc. A reopening of the fistula is thereby necessitated, which is allowed to persist for a few weeks more, and is then closed again and allowed to remain closed as long as the patient remains free from pressure symptoms.

6. Postoperative Fever.—Postoperative fever may be considerable after intracranial operations without being due to sepsis. Whether these higher temperatures are "absorption fevers," due to taking up by the circulation of the products of broken-down brain tissue, or to pressure on or trauma to some central "heat centre," it is difficult to say. At any rate, a temperature of 103° to 104° F., for a day or two after a clean operation, does not mean that the wound must be reopened, or that the patient is necessarily in a serious condition. Temperatures of 101° to 102° F. are not infrequent during the first few days following operations.

Necrosis of the bone flap after osteoplastic operations is occasionally a factor, probably, in these postoperative rises in temperature, though by itself it is not capable of producing anything above a low grade of fever.

Necrosis *in toto* or in magna parte of the bone flap is unusual and can only occur when the bone is widely denuded of its periosteum, or when in the absence of flap forceps to hold the osteoperiosteal flap to the galea, manipulation of the tissues causes separation of the galea from the bone, something that is likely to happen to any operator unless his assistants have been trained to look out and guard against it, or when suppuration is present.

More frequent, but still unusual, is a slight discharge of particles of bone and sand when edges of the bone flap have undergone a slight necrosis.

But, in general, bone necrosis is a very slight danger, and it has been known for many years that isolated bone fragments may be safely reimplanted when infection and undue pressure are absent.

7. Effects of Excision of the Cortex.—The after effects of cortical excision will depend chiefly on their location and extent. When occurring in the sensorimotor areas they will leave functional defects behind them. When occurring in the so-called silent areas they usually will not. When the excision of cortex is not combined with tumor removal or other manipulation of the brain, as is typically the case in the treatment of Jacksonian epilepsy, the defect will be a more or less sharply defined one, varying in size and character with the extent and location of the excision. Since the excisions for Jacksonian epilepsy are usually done in the motor areas these defects will usually be motor defects. If the excised area be small the defect will be small, as a rule, and tend to become speedily compensated. Larger excisions produce larger defects, which tend to be compensated more slowly, if at all.

In the cases in which there are preëxisting gross lesions of the cortex, which are removed at the operation, such, for example, as porencephaly, cortical necrosis, tumors, and the like, only relatively small additional defects may appear after operation, even though the amount of excised tissue may have been large.

The full effect of the excision does not develop at once, but gradually, as a rule, in the course of a few hours. This fact may be especially well demonstrated in the cases of cortical excision which are done as the second step of a two-step operation, the second step without general anesthesia. In these cases, the patient remaining fully conscious all the time, the gradual increase of the defect in the few hours following the operation may often be very definitely demonstrated.

If the lesion be in the motor area, a hypotonic paralysis will be produced, which gradually increases in extent and severity for the first few hours, and is then relatively rapidly followed by a spastic paralysis, which gradually increases on the succeeding days or weeks. If the excised area be small, these local defects may be gradually compensated in the course of a few days or weeks, as the case may be. When the excision has been extensive, however, there is much less tendency to compensation, and if it does occur at all, it takes a much longer time. In the cases in which the excision has been done in the lower part of the left motor area, there is great danger of Broca's motor speech area being involved, with consequent motor aphasia. There have already been enough of these unfortunate cases, either operative accidents or excisions necessitated by Jacksonian attacks, beginning in the lips, tongue, or about the mouth, so that we must regard Marie's contention for the non-existence of the pure types of aphasia as definitely overthrown. It now can be stated that pure lesions of Broca's area produce pure motor aphasia, and of Wernicke's area pure sensory aphasia. The lesions of Broca's area are very apt to be combined with agraphia from involvement of the adjacent hand and finger centre; and the lesions of Wernicke's area are likely to be accompanied by alexia, if at the same time the connections with the visual area in the occipital lobe be interrupted.

The defects after *excision of the sensory areas* are usually less marked than those after the motor excision, and are usually more quickly compensated. The disturbances may vary from slight astereognosis to complete sensory paralysis, but the lesser degrees of sensory disturbances are the rule. The patients notice and complain of these defects much less than they do of those of motility.

Occasionally *distant symptoms* are produced by the apparent involvement of regions away from the part excised. These distant effects are usually explained as the effect of the cutting of commissural, projection, or association fibers in the excision of the cortex, and this effect on distant regions by acute local processes has received from von Monakow the name of diaschisis. On a somewhat similar basis may be explained the sudden improvements and recoveries which sometimes occur in patients who, for some days or weeks, showing large and appar-

ently hopeless defects, then convalesce with surprising rapidity. Probably such symptoms are largely the result of the "local shock" of the operation.

In some cases of extensive cortical excision of large extent spastic contractures may ultimately develop, especially after the patient has escaped from continuous medical observations. These contractures, in my experience, may always be avoided by proper mechanical and orthopedic measures, if the patient remains continuously under medical observation. They are much easier to prevent in advance than to cure once they have occurred. The treatment of deformities, the result of neurological lesions, is a wide field in itself and lies beyond the scope of our present article.

When the cortical excision has been done for tumor, granuloma, or other local lesion, and particularly when there are local defects before the operation, these local defects will usually become much more marked after the operation, and are more apt to remain as permanent defects than are those of simple excision of the cortex, as for Jacksonian epilepsy. A continued increase of the defect even after months have elapsed speaks most positively for the continued growth of the tumor. Before that time such defects are usually more properly ascribed to local circulatory disturbances, such as edema and softening, or to diaschisis.

BRAIN PUNCTURE

While our present conception of the technique and clinical value of brain puncture dates from the publication of the thorough experimental and clinical study of Neisser and Pollock, yet Middledorpf, in 1856, had suggested the procedure on the basis of experimental evidence. Maas, not long afterward, insisted on its use as a preliminary step in the operative treatment of brain abscess.

In this country Souchon, in 1889, developed a technique from animal experimentation. Schmidt, four years later, pointed out the surgical possibilities of the procedure, particularly in connection with the treatment of abscesses of otogenic origin.

Payr, a little later, extended the technique to include the harpooning of brain specimens for histological examinations, a procedure which has found little favor elsewhere, and justly so, on account of the very considerable dangers and relatively small benefits connected with it.

The Kocher clinic, as early as in 1899, had a well-developed technique, which was published by Albert Kocher, and does not differ particularly from that at present in use.

One year before Neisser and Pollock published their work, Nicholas Senn published a case of internal hydrocephalus, which he had treated by connecting the subcutaneous tissue of the scalp with the ventricle by means of a thin rubber drain, using at the same time elastic compression of the skull. This method of ventricle drainage was first used by G. A. Sutherland and Watson Cheyne (who used folded lengths

of catgut instead of rubber), and later, though published earlier, by Henle, who used a gold tube.

Since the appearance of the paper of Neisser and Pollock, the method has been tried extensively and has come to occupy a permanent, if rather limited, field of surgical usefulness.

B. Pfeifer has reported the experience of Wernicke's clinic at Halle, and F. Krause has reported his experience on a still larger material in Berlin.

Indications for Cerebral Puncture.—For diagnostic purposes brain puncture is at present done chiefly after the dura has been exposed by cranial resection, so that possible hemorrhage may be controlled and purulent fluid be walled off from the meningeal spaces. It is generally employed: (1) to locate subdural and epidural hematomata; (2) to locate collections of pus; (3) to locate cysts of various origins; (4) to locate and determine the character of brain tumors—a dangerous and not very reliable procedure, but still used by F. Krause.

For the purposes of treatment it is used for (1) temporary and permanent drainage of the ventricles in internal hydrocephalus, acute and chronic, whatever the origin; (2) evacuation of pus and blood; (3) evacuation of fluid from meningeal space and lateral ventricles to diminish pressure before decompression or exploratory operations.

Dangers of Brain Puncture.—1. The chief danger is from hemorrhage, which is naturally very much less when the puncture is done in connection with a resection of the cranium, so that large veins can be avoided and any bleeding promptly seen and checked.

In the use of the puncture through a small trephine opening by the method of Neisser and Pollock, the chief danger is from injury to the pial veins by the point of the exploratory needle. Though normally small, in diseased conditions they are often greatly dilated and the walls are at all times very fragile and delicate.

The dural veins are injured less easily, but may give rise to very severe hemorrhage at times.

The branches of the middle meningeal artery may be injured during the trephining, but are more easily seen and the hemorrhage controlled by ligation.

Vascular tumors on the surface and in the tissue of the brain are still more dangerous to puncture, *e. g.*, angiomas, angiosarcomata, etc., may exist, which may easily give rise to fatal and unrecognizable hemorrhage.

2. Septic infection is rarely likely to be carried in from without, but may easily infect meninges from a punctured abscess if the Neisser and Pollock puncture is done without walling off the meningeal space.

3. Tumor infection may easily be carried into the healthy parts of the brain by the exploring needle when the tumor lies superficially and the needle, as often happens, is inserted through and beyond it.

4. Focal symptoms from destruction of nerve centres are avoided by not putting in the needle too deeply (four inches is the limit in any direction, and 5 to 6 centimeters are usually far enough).

Pons, medulla, and basal ganglia lie so deeply that there is rarely danger of striking them. Puncture in the motor area may have disagreeable after-effects, as in a case of F. Krause's, which was followed by temporary arm paralysis and severe general prostration and shock.

5. Sudden collapse from diminution of intravenous pressure may occur with the larger collections of blood and pus, but it is especially apt to occur in the evacuation of an internal hydrocephalus. If this happens the head is lowered and the veins filled with salt solution and adrenalin. The best means of preventing this complication is slowly to evacuate the fluid.

6. Acute and fatal edema of the brain associated with great increase of intracranial pressure occasionally follows puncture of the ventricles. We have seen alarming symptoms develop in two cases operated on by us when exploration was being carried out. Repeated emptying of the lateral ventricles and stimulation restored the patient.

Technique and Location of Brain Punctures.—There are a few obvious rules to be observed which makes the location of the puncture a relatively simple performance. (1) Avoid bloodvessels. (a) Punctures are not made near the midline nor near any of the venous sinuses, for fear of wounding the larger bloodvessels as well as the sinuses themselves (*i. e.*, transverse sinus, longitudinal sinus, sigmoid sinus, occipital sinus, etc.) (b) Also to avoid the neighborhood of the larger bloodvessels, particularly the middle meningeal artery. (c) In general, avoid puncturing through a small opening because of the difficulty of seeing what one is puncturing and of recognizing hemorrhage promptly when it occurs. (d) Do not puncture through a sulcus, but through the convexity of a convolution, because the vessels run in the sulci.

2. Explore for pus through the lowest possible openings, thus securing the best possible drainage if pus be reached.

3. Tap hematomata from the point of visible injury (unless there are other focal symptoms and the visible injury is slight).

Technique of Simple Puncture.—The locality of the puncture is shaved, washed with soap and gauze, alcohol and bichloride, 1 to 1000. Local anesthesia is usually sufficient, but an anesthetist should be at hand and all preparations, as a rule, should be made for a flap operation, if it becomes necessary. (a) Freezing with ethyl chloride, or (b) infiltration with Schleich's solution (No. 1), or novocain ($\frac{1}{2}$ per cent. with 12 m. of adrenalin, 1 per cent. to the 100 c.c.) may be employed.

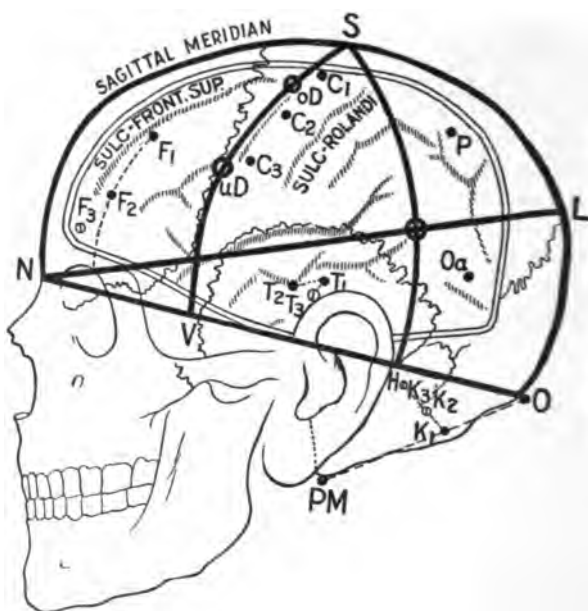
A small semicircular scalp flap may be made or the drill may be pushed directly through the scalp and set at once to work on the bone. If De Vibbiss' trephine is to be used it is best to make a small flap.

The drilling through the bone should then be done in the direction in which the needle is later to be inserted rather than at right angles to the bone. A metal shoe may be attached about the shaft of the drill (1 to 1.5 cm.) so that it will not suddenly penetrate the bone and lacerate the brain beneath. The position of the shoe may be generally changed as the drill goes farther in. When the bone has been penetrated the needle should be inserted, after the drill is removed, and the scalp

should be held fast, free of the bone, at each side of the opening by an assistant.

It may happen, especially in trephining through thick, fleshy parts, or especially in fat parts, as in the occipital or temporal regions, that it becomes impossible to locate the small trephine opening with the needle point, thus necessitating trephining anew. The needle should be about 6 inches long, graded in centimeters, no thicker than 1.8 mm., and provided with a mandrin. The needle should be attached to an aspirating syringe of about 5 c.c. (1 to 10 c.c.) capacity, and provided with a glass cylinder, so that the fluid can be seen, if aspirated. The tip of the needle is then inserted through the dura and gently advanced in the desired direction up to a depth of 5 or 6 centimeters, depending on location.

FIG. 81



Cranial puncture points. (Neisser and Pollock.) Heavy black lines are the craniometer lines. Solid black points indicate puncture areas of the different lobes. Shaded points for puncturing abscesses. *F*₁, *F*₂, *F*₃, puncture points for frontal lobe; *T*₁, *T*₂, *T*₃, puncture points for temporal lobe; *Occ*, puncture point for occipital lobe; *P*, puncture point for parietal lobe; *K*, *K*, *K*, puncture point for cerebellum lobe; *P*, *M*, mastoid process; *O*, ext. occip. protuberance; *N*, nasion (sabella); *S*, *V*, line of direction of precentral sulcus (anterior diagonal meridian); *S*, *H*, posterior diagonal meridian; *N*, *O*, base line; *N*, *L*, nasolambdoid line; *O*, *D*, juncture of upper and middle third of the central gyrus; *N*, *D*, juncture of lower and middle third of the central gyrus.

If no fluid appears the aspirating syringe may be attached and gentle suction exerted as the syringe is withdrawn.

If fluid is not found, the needle is cleaned out with the mandrin and then inserted in a new direction. It should never be moved into a different area of the brain without first withdrawing it entirely and also examining its patency.

When the examination has been completed the wound is washed off with bichloride, 1 to 1000, and alcohol, and then sealed with collodion and cotton. A stitch or two may be necessary, depending on the size of the opening in the scalp.

Puncture of the Ventricles by Way of the Corpus Callosum.—The object of this procedure is to establish a permanent connection between the ventricles and the subdural space accompanied with as little damage as possible to the brain and without having to leave a foreign body in the form of a tube or drainage wicking in the brain substance. At first von Bramann made a flap, 4 centimeters square, beginning 1 centimeter to the right of the sagittal suture, and as far from the frontal suture. The dura was turned to the side as a flap. The arachnoid veins emptying into the longitudinal sinus on the right side were then tied off and the right hemisphere separated from the falx. A sound-tipped cannula was introduced along the falx in the direction of the precentral sulcus. Then the corpus callosum was perforated and the sound introduced further, until liquor made its escape. By moving the cannula forward and backward from 1 to 1½ centimeters, a slit was made in the corpus callosum and then the sound removed, the dura closed, and bone and skin flap replaced and sutured.

Since his first publication, Anton has simplified his procedure. He makes an incision 3 centimeters long, somewhat behind the bregma, and a little to the right of the middle line. The periosteum is freed and the skull opened with a large Doyen burr. The sound is introduced through a small hole in the dura and passed between the brain and the dura until it meets the falx. Then it is passed along the falx until it perforates the corpus callosum. The procedure is then completed, as above. In case the large fontanelle is still open, as is so often the case in hydrocephalus, this opening is used. At its lateral border an incision is made through the skin and periosteum, the dura is opened in a non-vascular place, and the procedure completed as above.

Hans Willige, Anton's assistant, has still further simplified the procedure by using Pfeiffer's technique for brain puncture. A thin steel burr driven by an electromotor, is pushed through the skin and galea. The motor is started and the skull perforated by using very slight pressure. Then the sound is inserted under the dura and along the falx as usual. The use of a De Vilbiss drill should make the procedure still simpler and easier and more aseptic.

The aim of this procedure of Anton's is to allow collections of fluid in the ventricles to escape into the subarachnoid space, where they will usually be passed on further or absorbed. That this absorption or escape does not always occur, however, is shown by the case of Krause. Anton recommends his procedure in all cases of acute and chronic hydrocephalus, including those complicating epilepsy and brain tumor. In the first 22 cases operated by Bramann and reported by Anton, 8 were for infantile hydrocephalus, 11 for tumors, 1 cysticercus, and 1 tower skull. There was no death in the series. The favorable results obtained were: Improvement of vision and improvement of the choked

disk, disappearance of headache, vomiting, and vertigo. The removal of the complicating symptoms due to the hydrocephalus allowed tumor symptoms to make themselves better manifest, and in some cases made possible their correct localization and removal. Anton also recommends the use of the sound to determine the size of the ventricles and the consistency of their walls, certainly a ticklish and uncertain procedure. He also recommends it for secondary hydrocephalus after intracranial operations when the prolapse is threatening to tear open the wound.

A later publication contains a report of fifteen additional cases with good tables, showing the results. Other operators do not seem as yet to have interested themselves much in the procedure.

BRAIN TUMOR TREATMENT OTHER THAN SURGICAL

Before leaving the subject of the treatment of brain tumors, mention should be made of some of the non-surgical measures which have of late been advocated for the cure of malignant tumors.

The most recent and also the most promising of the attempts to find a medical treatment is that of Wassermann. After a long series of experiments he finally discovered in the salts of selenium and tellurium substances which, when injected into the blood stream, or into the tumor itself, produce a necrosis of the tumor. The best combination so far used is the eosinate of selenium. The dose is $2\frac{1}{2}$ milligrams for a mouse of 15 grams' weight. This dose is given daily for four days. At the end of this time the tumor becomes soft and fluctuating. Two to four injections are given later in order to insure the complete liquefaction and absorption of the tumor. The mouse cancers thus cured over several months' time were: Three varieties of the Jensen type; one type of Schilling's, one of Ehrlich's, and two tumors occurring spontaneously in the laboratory animals. Unfortunately the large doses of the drug given as above, and necessary for the curing of the cancer, were in many instances fatal to the mice, and especially to those mice which were already cachectic from advanced cancer. According to von Hansemann the action of the drug is on the nuclei of the cells for which it has a special chemical affinity. These show early pyknosis and disintegration. The rest of the cell then dies as a result of the death of the nucleus. No leukocytes invade the liquefied masses unless there is secondary infection. The result of the increased absorption of chromatin was seen especially marked in the spleen, and somewhat less in the liver.

This work of the Wassermann school is still too recent to have received confirmation at the hands of other workers. It does, however, seem to offer more hope of success in this line than anything that has been suggested since Hodenpyl's experiment at Roosevelt Hospital. Perhaps it may not turn out to be such another disappointment. It must be borne in mind, however, that it is one thing to cure cancer in the mouse and quite another to cure it in the human being; and also that any drug

producing as many fatalities in man as this drug does at present in mice could never come into very general use. Loeb, of St. Louis, claims similar successes by the use of colloidal copper injections.

Various other more or less toxic substances have been recommended or used. Von Dungern has tried *crotalus* (snake poison) on rat sarcoma, with favorable results.

Coley's mixed toxins for the treatment of sarcoma are well known, and have made a place for themselves in the treatment of inoperable sarcoma, which the profession generally recognizes. He has recently reported sixty-five cures of his own, and thirty in the hands of other authors. These cures vary in length from three to eighteen years.

Adamkiewicz's cancrudin and Schmidt's antimeristem have also achieved some foreign reputation in the treatment of cancer. Two recent papers report results with the latter.

X-rays, radium, and fulguration practically do not come into consideration in the treatment of intracranial tumors.

Czerny and Caan and lately Noehte have used salvarsan in the same connection, but without any really gratifying results.

The entire subject of experimental cancer treatment has been lately reviewed by C. O. Jensen, to whose paper may be referred those who want more details of this recent work and a more comprehensive literature.

SURGERY OF THE SPINAL CORD

The spinal cord presents difficulties of attack scarcely less than in the case of the brain, owing to its deep-lying bony box. The prospects of operative success are, however, somewhat better because of the more accurate topical diagnosis here possible; but the possibility of complete recovery is somewhat less because of the delicate construction of the cord and because none of the areas injured are "silent areas."

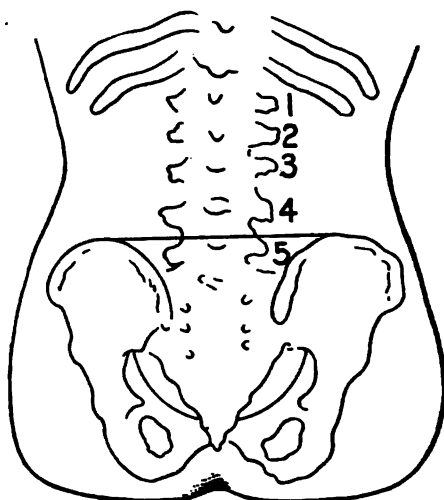
Spinal Puncture.—Although the chemical and physical characters of the spinal fluid in health and disease, its origin and movements, have been studied in animals and man since Cotugno's time (1769), it is only of recent years (Quincke, 1891) that we have been able to use it for diagnosis and therapy.

Indications.—The chief indication for the performance of lumbar puncture occurs in connection with eye symptoms is meningitis in its various forms. Here the puncture is an invaluable diagnostic aid and likewise an important aid to therapy. All other indications are very infrequent when compared with meningitis, the presence of which can be diagnosticated and its varieties differentiated by examination of the spinal fluid obtained.

Drainage of pus and irrigation, to a slight extent, may be practised by means of the puncture, but much more important, from a therapeutic standpoint, is the injection of the Flexner serum into the subarachnoid space in cases of epidemic meningitis.

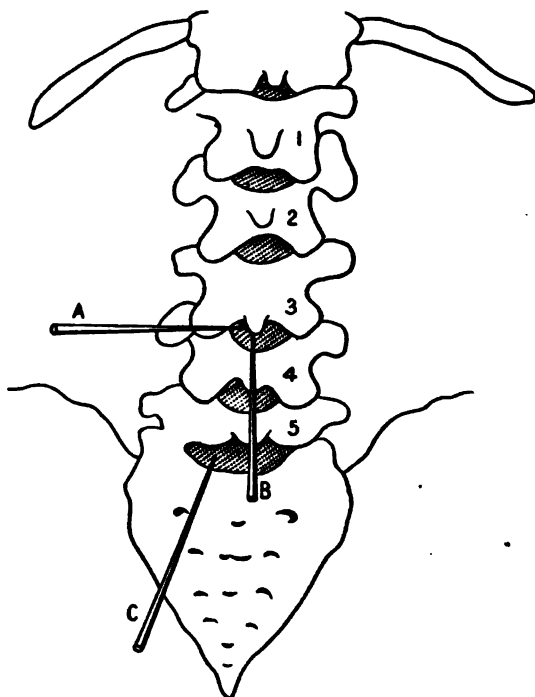
Intrameningeal hemorrhage may also be diagnosticated by the

FIG. 82



Spinal puncture. Relations between the lumbar spines and crests of the ilia shown by the transverse line.

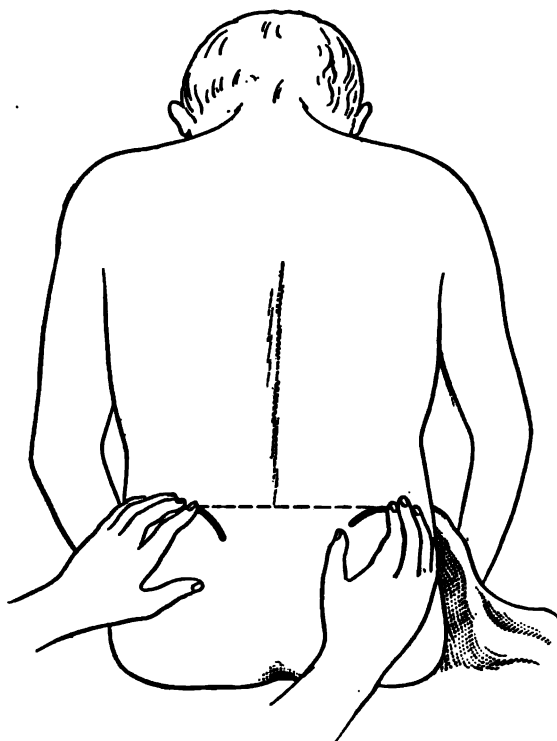
FIG. 83



Method of puncture for spinal drainage. A, Quincke's site; B, Marfan's site; C, Chipault's site.

puncture, but always has an element of doubt, inasmuch as a vessel wounded by the needle may lead to a similar finding, although in the latter instance the blood, as a rule, is redder than in the former, and less diffusely distributed through the fluid, being usually more marked in the first few drops than in the last.

FIG. 84



Topographical relations for spinal puncture, according to Tuffier. The line joining the crests of the ilia passes directly over the spinous process of the fourth lumbar vertebra. The needle is inserted just above the spinous process to reach the third interspace, or below it to reach the fourth.

Increased intracranial pressure, whether due to tumor cerebri, hydrocephalus, hemorrhage or meningitis, may often be diagnosed by the increased pressure of the spinal fluid, which, normally flowing 60 m. per minute from the needle, may fairly spurt out when pressure is high. The normal pressure is 5 to 7.5 mm. of mercury, or 60 to 100 mm. water, which may be increased under pathological conditions to 15 to 60 mm. of mercury, or 200 to 800 mm. of water.

In the last few years the cytological, chemical, and biochemical examinations of the spinal fluid, as obtained by spinal puncture, have assumed great importance in the diagnosis of syphilis and parasyphilides.

The mononuclear leukocytosis, excessive presence of globulin, the Wassermann and Noguchi tests have made the differentiation of the

parasyphilides from syphilis, and both from non-syphilitic affections, much more accurate.

Other less important indications are derived from the presence in the spinal fluid of an excess of urea in uremia, and the presence of cholesterolin and cholin in cases of various degenerative process of the central nervous system.

Therapeutic Indications of Spinal Puncture.—The application of therapy by means of spinal puncture is always associated with danger to life. The evacuation of fluid, whether it be normal, purulent, or hemorrhagic, always entails some risk of allowing the medulla to sink into the foramen magnum and become incarcerated there, to the detriment of its vascular supply. The central canal and subarachnoid space may also become blocked, thus leading to an acute hydrocephalus or pyocephalus, as the case may be.

To avoid this danger only a small amount of fluid, usually 20 to 40 c.c., should be evacuated at one time, and the patient should be placed in the lateral and *not* in the sitting position during the procedure.

The evacuation of the pus in meningitis by spinal puncture is not of much therapeutic value. Puncture is of importance here chiefly because it enables us to introduce Flexner's antimeningococcic serum directly into the subarachnoid space. For this serum injection the needle is introduced in the ordinary way, a few drops of fluid allowed to escape, and then the serum is injected through the needle 20 to 100 c.c. The procedure may be repeated every day until convalescence begins. The unanimous enthusiasm of clinicians over the whole world proves the great value of this procedure. By it the mortality of the scourge has been reduced from 75 to 25 per cent. whenever it has been used. It is a great discovery of which America may well be as proud as France is of Behring's serum.

The treatment of increased intracranial tension by lumbar puncture had considerable vogue to within the last few years. Cushing has laid such emphasis, however, on the danger of medullary hernia into the foramen magnum in such cases, and there have been so many such cases reported, that it would hardly be justifiable at the present writing to adopt such a procedure, in view of the excellent results now obtained by cranial procedures.

Technique of Spinal Puncture.—Quincke recommended that the puncture be made between the second and third, or third and fourth vertebræ. I nearly always use the latter location because of the readiness with which it is found by drawing a line between the crests of the ilia. This line passes over the fourth lumbar spinous process and just above it I insert the needle.

Many clinicians recommend that the puncture be made in the mid-line. I prefer, however, to enter the needle one-half to one centimeter to one side of the median line, as originally proposed by Quincke, in order to avoid going through the tough ligamentum interspinosum.

The needle can also be inserted between the fourth and fifth vertebræ or between the fifth vertebra and the sacrum, and it is not improbable

that in an occasional instance pathological constituents of the spinal fluid, which have settled to the lowermost part of the subarachnoid space, may be found here, though missed on puncture at the usual location. Such an occurrence, however, is certainly very rare and is not a practical consideration.

FIG. 85

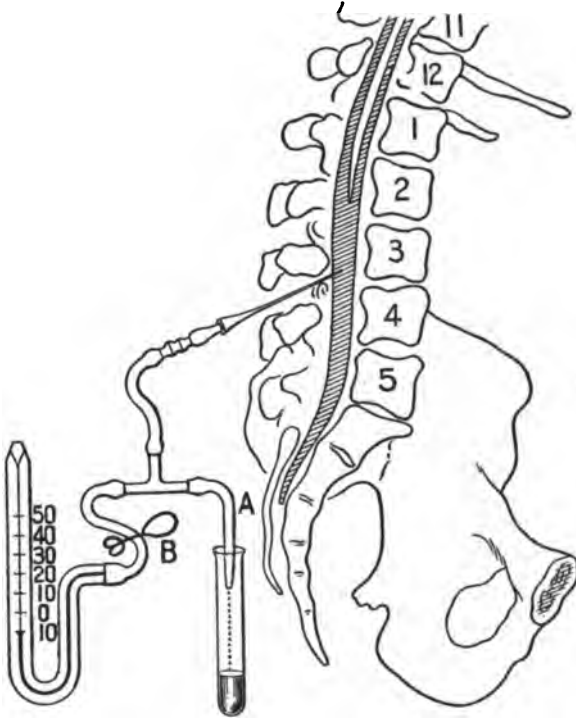


Diagram of lumbar puncture. T-shaped glass tube for combined drainage and estimation of the pressure of the spinal fluid.

The needle, once entered in the skin just outside the midline, is pushed inward, slightly upward, in adults, horizontally in children, and medianward. The dura is reached at a depth of four to six centimeters in adults, or of two centimeters in a two-year-old child. If the tip of the needle engages bone at the first attempt, withdraw a little, change the direction and try again.

The position of the patient during the puncture should be the lateral recumbent, with the spinal column flexed to a maximum, in order to enlarge the posterior intervertebral aperture.

I prefer the recumbent to the sitting posture because I believe that there is less danger of cerebral vascular disturbances or hernia of the medulla into the foramen magnum in the lying position.

When the spinal fluid runs out it is caught in a sterile test-tube.

The rate of flow and incidentally the pressure of the spinal fluid is determined by counting the drops as they flow from the end of the needle. The normal rate of flow is about 60 drops per minute. More than this number means increased spinal and consequently increased intracranial tension. Occasionally, when the pressure is very high, the fluid may flow in a continuous stream instead of in drops. A number of drops less than sixty per minute indicates either hypotension or partial plugging of the needle.

The needle should be withdrawn as soon as the pressure falls to normal. As a rule it is safe to stop somewhat before normal in view of the ever-present danger of hernia of the medulla into the foramen magnum.

It seems hardly necessary to state that the needles should be sterilized by boiling in 1 per cent. sodium carbonate solution, and the skin sterilized by some one of the accepted methods. Simple disinfection with 95 per cent. alcohol or painting on of tincture of iodine, according to Grossich, is sufficient.

An anesthetic of any sort is usually not necessary, although it may be desirable at times to anesthetize the skin with the ethyl chloride spray.

The examination of the spinal fluid is more appropriately discussed in one of the standard text-books on laboratory diagnosis, such as Webster's *Diagnostic Methods*, Blakiston, 1909; Wood's *Clinical Chemistry*, Appleton, 1905; Sahli's *Klinische Untersuchungs-Methoden*, F. Deuticke, Wien, 1909.

One of the more recent text-books is advisable, since otherwise the important new biochemical reactions of the spinal fluid, like the Wassermann, Noguchi, and Nonne-Apelt will be missing.

Technique of Laminectomy.—**Preparation of the Patient.**—Although the Grossich iodine preparation has given us a potent aid in emergency cases I do not yet feel certain enough of its efficiency to use it in other cases, save when a preliminary preparation has been done on the previous day. I lay great stress on having the tincture of iodine as fresh as possible when I do use it. It should not be over a week old and should always be made up according to the directions of the new pharmacopœia, that is, containing 7.5 per cent. of potassium iodide. Patients with spinal cord lesions are especially liable to develop trophic disturbances on pressure points, especially on the back; and these lesions may develop from bullæ which have arisen after the use of a too old tincture of iodine. This bulla formation is probably due to the hydriodic acid contained in the old tincture. This acid content may reach as high as 1 per cent. in some of the old tinctures, made according to the previous pharmacopœial formula, without potassium iodide. The action of the hydriodic acid on the skin is probably a little more severe than that of hydrochloric acid. In cases in which we use the iodine I always wash off the excess with alcohol as soon as the operation is completed and the skin sewed up.

Position of the Patient.—The patient is made to lie on the table face downward, with a sandbag under each shoulder and a soft cushion

under the abdomen, so as to arch the back somewhat. By thus raising the shoulders, thoracic respiration is left relatively free, and the heart action is not impeded by pressure against the table. The back is not quite so well arched in this posture as it is with the patient lying on his side, but by using a well-padded pillow under the abdomen there is generally no trouble in getting good operative access to the cord.

The advantages of this position over the lateral position are: (1) The patient lies more rigidly on the table, a matter of importance in fracture and dislocation cases; and it is easier to get him on and off the table without disturbing the alignment of the vertebræ than it is when the lateral position is used. (2) The operator and his assistants work more easily and advantageously than in the side position. It is also easier to secure good illumination in the bottom of the wound. (3) A plaster-of-Paris retaining splint applied to this position can be more accurately adjusted to the contour of the back, and will be more comfortable to the patient as he lies in bed.

Narcosis.—We always use ether by the drop method, often preceded, fifteen minutes before the operation, by a hypodermic of $\frac{1}{4}$ grain morphine and $\frac{1}{160}$ grain atropine.

The method of local anesthesia by 0.5 per cent. novocain and 1 per 1000 adrenalin (15 minims of adrenalin solution to 100 c.c. of novocain solution), which H. Braun originated and which is highly recommended by Prof. Bier and Krause, we mention only to condemn. It is out of line with practice in our country. Its use abroad we believe is due solely to the fact that Germans are practically unacquainted with the good routine use of ether by the drop method. With them general anesthesia usually means the use of the Billroth mixture, chloroform, or ether by the so-called "Ethernäusch" or suffocation method. The reason that spinal anesthesia and local anesthesia have so large a present vogue on the continent is due to the fact that the externes, usually junior and senior students who administer the general anesthetics, use with little experience the most dangerous anesthetics.

The Incision.—We always use the straight median incision. The flap method, with and without osteoplastic resection of the arches, we tried for a time some years ago. We found it a very different procedure on the living from what it is on a cadaver. Accurate reposition of the flap is usually impossible and healing is much slower than with the older and better technique. It is safe to state, we believe, that the great majority of prominent operators now use the longitudinal incision.

The spinous process, which is to be the middle of the incision, is marked at the last examination, before operation, with a silver nitrate stick, slightly moistened. To the mark thus made is applied a sponge, wet in photographic developer. Such a mark can be obliterated only by repeated scrubbing and is usually present for many days.

The length of the incision will depend, of course, on the number of arches to be resected. It is not necessary to make a long incision at first, because the longitudinal incision always admits of extension in

either direction. This is an advantage which the flap operation does not possess.

Operative Technique.—The wound is held apart by retractors. The fasciæ and muscles of the back are separated from the spinous processes down to the neural arches, using a large flat chisel in preference to a periosteotome. The separated muscles are promptly covered with towels wrung out of hot salt solution. Deep retractors applied over the towels hold the wound open. The heat and pressure soon control all hemorrhage from the sides of the wound without the necessity of applying any clamps or ligatures at all. The speed of the operation is thus greatly accelerated and the wound opening is not littered up with artery snaps, which obstruct the view and retard the operator. This use of towels or dressings, wrung out of hot salt solution, is our practice whenever an incision is made through large masses of muscle. It is indispensable for rapid bloodless work in the radical treatment of carcinoma of the breast, and but little less valuable in laminectomy. It is much preferable to tamponing the wound, as recommended by Krause, in that it stops hemorrhage fully as well as the tampon and necessitates no pause in the operation. The periosteum is next stripped from the posterior surfaces of the neural arches lateralward. If there is then a large enough space between the arches, we slip a DeVilbiss forceps, with an extra long shank, in between them, and cut them through just as we cut through the skull. When there is not room enough for the forceps we make a trephine opening through the arch with a Hudson or De Vilbiss burr. Through this trephine opening we insert the De Vilbiss forceps and cut the arches in both directions as far as necessary. The same procedure is then repeated on the opposite side. In the upper thoracic and in the sacral region we sometimes use the Doyen saw in place of the De Vilbiss forceps on account of the rapidity with which the arches may be divided by it in these regions. In the concave portions of the column, the cervical and lumbar regions, the use of the saw is not practicable.

The spinous processes thus cut free are separated from their connections above and below with the remaining spinous processes. They are loosened from the spinal dura by blunt force. The spinal dura then lies free in the bottom of the wound, covered by epidural fat and a plexus of small veins. These superficial structures are separated in the mid-line and pushed to either side.

The restoration of the bony arches by the replacement of the bone fragments does not appear to us essential. Therefore, no attempt is made at this stage to form an osteoplastic flap.

The dura is carefully inspected and palpated. Normally, it usually shows pulsation, either synchronous with respiration or with the pulse, or with both; but there are frequent normal exceptions.

If the exposure is not free enough at first the sides of the arches are removed with a rongeur forceps. It is, in fact, nearly always a good plan to cut away the arches practically up to the articular and transverse processes, thus opening the operative field to its maximum extent.

Tumors, blood clots, bone fragments, and pus are then dealt with according to methods which we shall discuss later under the title of Special Procedures.

If, however, the lesion be not discovered at once after the fullest possible exposure of the field our next recourse is to probe. A blunt and somewhat flattened flexible probe or a Braantz sound is used. The dura is gently separated from the bone, upward, then downward, and then anteriorly. Special care must be taken to avoid injuring the spinal roots. If any abnormal resistance be found, its location is exposed by further resection, inspected and palpated.

If external inspection and palpation give a negative result the dura must be opened. The opening is made by a longitudinal median incision, the dura being held up between blunt forceps, just as is the peritoneum when incised. Incision, once made, is extended the full length of the operation wound, using scissors with a grooved director as a guide. The cord is next carefully inspected and very gently palpated.

To minimize the loss of liquor in opening the dura we usually tuck simply a little gauze between the dura and the bone, at the upper end of the wound. By so doing the loss of liquor is greatly diminished and no damage done to the cord, nor does the tampon interfere materially with the intradural use of the exploring sound, since it may be readily dislodged if too tight. To pass a ligature extradurally entirely around the cord in order to prevent a loss of spinal fluid (Sick's procedure) we believe to be a dangerous undertaking. The nerve roots are jeopardized in the passing of the cord, and the integrity of the cord itself is threatened with the ligature tied, the degree of tautness necessary to stop the flow of liquor approaching dangerously near to that which overcompresses the cord. We have yet to see a case develop serious symptoms from loss of spinal fluid when the tampon alone was used.

If inspection and palpation of the cord does not reveal the sought-for lesion, our next step is to probe intradurally, upward, downward, and even anteriorly. At both sides of the cord the sound is apt to lodge in the ligamentum denticulatum, which must, therefore, be avoided and must not be confused with the resistance caused by a tumor or other lesions. At this stage of the operation it is often a valuable thing to have the indelible mark of silver nitrate on the skin. By reconstructing the spinous process which this marks, we have a valuable landmark always at hand, which may be no small help to us in searching for the lesion and in determining in which direction further operative exposure shall be made in case we fail to find the lesion in that section of cord already examined.

The opening of the dura must be carefully performed in order not to overlook a localized meningitis serosa. If this condition be present it will then be visible only as a thin, transparent membrane, bulging up through the incision in the dura. This membrane promptly collapses when incised, extruding the contained fluid, and is thereafter identified only with much difficulty. When a meningitis serosa is found the operator must not be satisfied, however, merely to have found it,

but must also look farther to find the cause for it, if possible, such as a tumor lying lower in the cord or dura and obstructing the passage of liquor, inflammation in the bone or dura, and the like.

Save in exceptional cases the lesion sought for will have been found when the operation has been carried thus far. If, however, the lesion is not found, failure will have been due to one of three causes: (1) the lesion lies inside the cord and causes no appreciable increase in the cord's bulk or consistence; (2) the diagnosis as to the localization or the character of the lesion is at fault; (3) a meningitis serosa has been overlooked by the operator, who, either on opening the dura, or subsequently on passing the probe, has perhaps noticed a sudden gush of fluid but nothing more. In case the lesion lies inside the cord the treatment to be followed varies according to its probable character. In general, if no change in the cord can be found on palpation and inspection, it is probable that the lesion, even if present, is not amenable to surgical treatment. In fact, many operators advise against any further attack on the cord.

In some cases, however, one or more longitudinal incisions, made into the cord at this juncture, have revealed the presence of a tumor or some other removable lesion. In some cases such tumors have been removed successfully, or have been extruded spontaneously after the incision into the cord. That such longitudinal incisions into the spinal cord, as a rule, do no serious injury to the structures of the cord is very probable from the work of A. R. Allen. In the use of such deep incisions as he makes (he even cuts clear through the cord in a longitudinal direction) we cannot concur. Such incisions, of necessity, must cut decussating fibers, if carried deeply, and will, therefore, produce symptoms easily demonstrable in man, even if they are not in the dog.

Postoperative Treatment.—When the lesion sought for has been found and removed, or otherwise treated, the wound is cleansed with normal salt solution and the dural opening closed with a continuous suture of fine cat-gut. The wound is again washed out with a normal salt solution and the fasciæ and muscles of the two sides are united with buried continuous sutures of catgut. The skin is sutured with silkworm gut, then cleansed with bichloride and alcohol. Tincture of benzoin is applied to the wound, allowed to dry, and silver foil is laid over it. On the top of the foil a few layers of gauze are placed and fastened with adhesive plaster strips. A combination dressing is laid over this and held in place with the binder.

When the plaster-of-Paris retention dressing is necessary, as in fracture and dislocation cases, sterile sheet wadding is laid over the combination dressing and covers the entire back, usually from the occiput to the middle of the buttocks. A moulded plaster-of-Paris dressing is then applied over the wadding, and is strengthened with reënforcing strips, especially over the neck and small of the back. This plaster bed is allowed to harden somewhat and the edges are trimmed up to fit comfortably to the patient. After the cast has dried fairly well, a coating of flexible collodion or shellac is applied, the patient is replaced

in the cast, which is bound firmly to him by roller bandages. He may then be turned over on his back, put on a cart, and taken back to his room. The cast is a great aid in subsequently moving and handling him, and protects him during these manipulations. In trimming it, allowance must, of course, be made for the use of the bed-pan. In cases in which the injury to the spinal column lies in the cervical region it is often well to envelop the whole head in the case, the dressing being applied while the head is in extension. The cast must then be closely fitted about the shoulders and pelvis in order to maintain this extension. A jury-mast attachment is best used when an entire body cast can be applied. Further details along this line may be obtained in any good orthopedic text-book.

In cases in which the bodies of the vertebræ and the transverse processes are intact no such retention dressing is, of course, necessary. The patient is simply put to bed on his back, is turned from time to time, and watched to prevent the development of bed-sores.

If the pulse be too rapid, if the patient seems to be in shock, or has lost much spinal fluid, normal salt solution is given hypodermically, or intravenously, as the case demands. We often add a drop of adrenalin to the pint of salt solution. Coffee and whisky are administered per enema, and perhaps a syringe of camphorated oil is injected under the skin. An ampulle of digipuratum, given by deep intramuscular injection, we have found especially valuable when cardiac insufficiency is present or threatening.

Complications and After-treatment.—As soon as possible after the patient returns to consciousness following operation, a careful examination of his motor and sensory functions should be made in order to determine whether there are any additional defects present as a result of the operation. In some cases this does occur, but, as a rule, the defect is slight and soon disappears. Its course should be observed carefully from day to day. If the lesion appears to progress a hemorrhage or other source of pressure on the cord must be thought of, and may sometimes make necessary a second operation. I have never personally had such a case, but there have been such reported.

Leakage of Cerebrospinal Fluid.—Leakage of cerebrospinal fluid is a more frequent complication, especially in the cases where the dura has to be resected, in consequence of its involvement in the disease process. The escaped spinal fluid may lie entirely under the skin, forming a soft fluctuating tumor, which must be differentiated from a hematoma. Such a collection of fluid is best removed by an aspirating needle and syringe, and as a rule, ceases to reform after one or two evacuations. In some cases, however, the fluid finds its way to the exterior through a suture puncture, or between the edges of the wound. In such cases the asepsis of the dressings must be scrupulously observed, and local applications to close the wound, such as silver nitrate stick, and the like, are to be avoided because of the danger of infection, which any small necrotic area, such as is always produced by the caustic, presents. As healing goes on the fistula gradually closes. It is no use, as a rule, to

run another suture around the opening. The results of such attempts is often to produce two leaks where previously there was only one.

We have the nurse on the case change the outer dressing as often as it becomes soaked through. We change all the dressings at least once daily, wipe off the wound with alcohol, and apply tincture of benzoin to it, followed by a dry dressing.

In cases in which it is possible to perform primary suture of the dura we rarely have any trouble. In cases in which it is necessary to resect the dura we put in one or two layers of buried sutures very tightly, thus preventing leakage of the fluid from below, and thereby nearly always avoiding this annoying complication.

The Prevention of Urinary Infection.—All our spinal cord cases receive 5 to 10 grains of urotropin three or four times daily, for a few days preceding the operation. When there are any bladder symptoms before operation, or when they develop afterward, we continue the urotropin treatment, as a rule, as long as they are present. The urotropin serves the double function of diminishing the danger of meningeal infection and of serving as a urinary antiseptic. In the cases in which there is urinary retention catheterization is performed twice daily under scrupulous asepsis. We always have the same interne do the catheterizing on a patient. Thus there can be no escaping of responsibility nor any sharing of it in case urinary infection develops. When infection occurs the bladder is washed out after each catheterizing once daily with a boric acid solution and once daily with silver nitrate, 1 to 2000.

The Prevention of Bed-sores.—This is chiefly a matter of careful nursing. If a patient has involuntary evacuations it is the nurse's duty to see that he is cleaned up immediately when one has occurred. The surgeon should find his patient clean at every visit. It is also permissible to find him being cleaned, but to find him soiled and unattended to should mean a change of nurses. Daily inspection by the surgeon of the sacrum and other points of pressure is necessary to insure good results. In case bed-sores threaten to develop, however, in spite of every precaution, then the only additional thing we can do is to put the patient on an air-bed or water-bed. When a cast has to be worn, the air or water is not practicable. In cases of complete severance of the cord it is especially difficult to prevent the development of bed-sores by any means at our disposal.

The Prevention of Contractures.—It is always possible to prevent contractures if seen in the beginning, and if the patient remain continuously under professional observation. It is not necessary to use apparatus to this end. This result is obtained by: Correcting and overcorrecting a tendency to contracture during daily massage; exercise devised to strengthen antagonistic muscles, in case there are such still present; electrical excitation of the opposing muscles.

Indications for Laminectomy.—These may be grouped in general into: 1. All those lesions amenable to surgical treatment which cause pressure on the cord and thereby interfere with its functions. These are:

(a) Tumors of the cord, membranes, and bone which can be removed at operation.

(b) Meningitis serosa circumscripta.

(c) Granulomata of the bone and dura.

(d) Fracture and dislocations of the vertebræ.

(e) Inflammatory processes of the dura and the bone.

Acute osteomyelitis.

Extradural abscess.

(f) Meningitis fibrosa chronica.

(g) Hematomyelia—hematorrhachis is better treated by spinal puncture.

2. Resection of the spinal nerve roots for contractures or for pains.

A. Resection of the posterior roots (the Dana-Foerster operation).

This is the more usual procedure on the spinal nerve roots. The reflex arc is thereby interrupted and spasticity diminished, a better result being obtained, as a rule, in the legs than in the arms. Or the roots may be resected to relieve the pain in gastric crises, or in obstinate regional neuralgias, by thus interrupting the sensory paths.

B. The anterior roots are rarely attacked, because of their relative inaccessibility and the danger of producing paralysis. They might, however, be cut to diminish contractures, provided not more than two, or at the most three, consecutive roots are cut. Cutting more than that number would most likely substitute paralysis for contracture.

3. For exploration and decompression:

When a malignant tumor is present, or some other source of pressure on the cord is suspected, which on operation turns out not to be removable, the operation may be termed exploratory and the operation may have, in the decompression, a satisfactory result. The pressure on the cord is thereby at least temporarily relieved. After stab wounds of the cord, when it is suspected that part of the symptoms are due to the pressure of a blood-clot, a similar exploratory operation may be permissible. The operation does not materially weaken the spinal column, and the removal of pressure from a softened, edematous, or hemorrhagic cord may do some good.

In general it may be stated that the operation of laminectomy is indicated whenever we have a lesion compressing the spinal cord, practically irrespective of its character—since that is a point very difficult to accurately determine—and provided that its location can be diagnosticated with a fair degree of certainty. In such cases it is practically useless to give either a short or a prolonged course of treatment with mercury or any other specific. If the Wassermann reaction be negative, operation is clearly indicated. If it be positive there is very little use in trying anything more than a short course of treatment. For the positive Wassermann reaction does not show that the *cord* lesion is syphilitic; and even if it be syphilitic, irreparable damage may be done to the cord before the specific drugs begin to act, just as is the case often with gumma of the brain. In case, however, the patient comes to us so early that the segmental diagnosis is not possible, then the

interval of waiting may be utilized for a course of antiluetic treatment. As soon as a reasonable suspicion as to the localization of a tumor exists, recourse should be had immediately to operation. In case the tumor be not directly cut down upon, the extension of the operation and the use of the sound, both outside and inside the dura, will usually enable us to locate it. It is but little satisfaction waiting until the patient is badly crippled before moving to his relief.

The differential diagnosis between intra- and extramedullary tumors is usually impossible. To wait until the diagnosis can be refined is not only useless as a rule but actually harmful. We need to know only the level of the tumor; further local diagnosis is readily made at the operation.

SPECIAL PROCEDURES FOR SPECIAL INDICATIONS IN SPINAL SURGERY

Tumors.—Aside from traumatic lesions the various forms of tumor, when including localized meningitis serosa, constitute the most frequent indication for laminectomy.

Tumors of the Bone.—In a well-taken x-ray picture these tumors may be frequently diagnosed. The treatment depends naturally on the character of the tumor.

BENIGN TUMORS of the bone are relatively rarely a cause for laminectomy. They are either osteomata or chondromata, which project into the canal and therefore have to be excised. When they occur at the sides of the cord or in the posterior part of the canal they are relatively easy to attack. When they involve the body of the vertebræ and are therefore located anteriorly they are difficult to get at without injuring the cord. The mallet, chisel, and rongeur forceps are the instruments employed in the removal of such tumors.

In attacking tumors of the vertebræ, so located that it is necessary to pull the cord to one side, the spinal nerve roots are often in the way. It must be remembered that at least three adjacent spinal nerve roots must be cut in order to produce a demonstrable defect.

Advantage of this fact may be taken to cut roots in preference to pulling or crushing them, or to using dangerous pressure on the cord in order to get it out of the way during the manipulations necessary for the removal of the tumor.

Some operators advise against attacking tumors lying in the bone anterior to the cord, because of the difficulty in reaching them without causing serious damage to the cord. It must be remembered in this connection that laminectomy will, of itself, largely serve to diminish the pressure of such tumors on the cord by its decompressive action. It is better, therefore, to fail to remove a tumor lying in a dangerous locality than to cause a serious transverse lesion of the cord in trying to get at it.

MALIGNANT TUMORS.—Malignant tumors are rarely helped by operation. They are best let alone. Support should be provided for the

spinal column in the form of a plaster-of-Paris dressing or orthopedic jacket, in cases in which these tumors produce marked weakening of the column. The operation is likely only to hasten the inevitable end. These tumors are usually metastatic epithelial tumors, chiefly carcinoma. A careful general examination of the patient and an investigation into the findings at previous operations for suspicious growths, together with an x-ray examination, will usually enable us to avoid the mistake of attacking, operatively, one of these unfortunate conditions. In cases, however, of metastases of the ossifying type, which usually fail to show in the x-ray picture, and which may have their origin beyond our diagnostic reach in the internal organs, we may occasionally be drawn into error. Fortunately for us, however, this ossifying type does not often produce spinal cord symptoms, because it does not soften the bone and rarely penetrates the canal.

Tumors of the Dura.—The method of using a sound to locate these tumors when they do not at once appear in the field of vision has been previously considered.

When a considerable portion of the dura has to be excised to remove the tumor a little packing should be put between the dura and the bone at the upper end of the wound in order to diminish the loss of cerebrospinal fluid. It must not be forgotten that a tumor lying outside the dura does not exclude the simultaneous presence of one or more lying inside the dura. A localized meningitis serosa is by no means a rare intradural complication of an extradural tumor. It is, therefore, a good rule to open the dura in all these cases and inspect the inner surface of the dura and the cord.

In closing the wound after the excision of a generous piece of dura, the deep catgut-sutures which unite fascia and muscle on the two sides, should be applied tightly in order to avoid leakage of cerebrospinal fluid. There is always plenty of periosteum present so that a plastic covering of the defect could be obtained, as a rule, if desired. But that means more handling of the tissue; bone might possibly develop after the operation, and our results to date have been satisfactory without it.

Tumors Inside the Dura.—When the exposure of the dura fails to reveal the presence of a tumor, a longitudinal incision should be made in the dura in the middle line for the purpose of exposing the subdural space and the cord. In making this incision the possibility of the presence of a localized meningitis serosa must be thought of. If the incision in the dura be made carelessly the cut may easily go clear through the filmy sac of the meningitis serosa, a slight gush of fluid takes place, and thus the exploration fails to show any change to account for the patient's symptoms. To avoid overlooking a meningitis serosa the dura should be picked up on each side of the middle line with smooth tissue forceps, and then incised just as is the peritoneum in a laparotomy. If a meningitis serosa be present its thin diaphanous wall will then be seen bulging out in the dural opening. If the tumor does not lie directly under the eye after the dura is opened, an exploration with the sound must then be undertaken, just as it was outside the dura. Special care

is necessary to avoid injury to the nerve roots or overstretching or abrading the cord. The ligamentum denticulatum, lying at either side of the cord, may easily be impinged upon by the sound, and must not be mistaken for a tumor. If the tumor found lies superficially in a groove in the cord it is removed immediately, but if it lies in the body of the cord, a longitudinal incision may be made into the cord over it in the hope that spontaneous extrusion may later occur by intramedullary pressure. The tumor may be removed subsequently at a second-step operation.

Hematomyelia.—The sudden onset of this condition, together with the history of trauma, often enables us to make a fairly probable diagnosis of the condition. It is differentiated from hematorrhachis by the absence of blood in the spinal fluid as obtained by lumbar puncture. Of course, hematorrhachis and hematomyelia may exist together, but this usually occurs only in the severe traumatic cases. Here lumbar puncture rather than laminectomy is the proper treatment, except where it may be necessary to remove bony fragments which are pressing on the cord.

Hematomyelia may be due to a hemorrhage into a glioma, angioma, sarcoma, or other tumor, and may be the first symptom to call attention to the new growth. It may be also the principal change in an acute myelitis, as in a case seen recently with Dr. E. P. Cook, of Mendota.

We do not recommend operation for uncomplicated hematomyelia, but in case it be found at operation the cord may simply be incised longitudinally and the accumulated blood permitted to escape.

Hematomyelia, however, does not in any way contraindicate operation. In case the diagnosis lies between a hematomyelia and some condition amenable to operative treatment, laminectomy should be performed. If hematomyelia is present the patient may be somewhat improved by the operation.

Meningitis Fibrosa Chronica (or pachymeningitis cervicalis hypertrophica).—This condition may occasionally be found when a tumor was diagnosed. When this thickened membrane is closely applied to the dura it may be excised; and even if rather extensive, the dural defect may be compensated for by using periosteum or fascia or merely uniting the muscles fairly closely about the cord. In the cases, however, in which the membrane is applied to the pia and perhaps to the dura at the same time, thus obliterating the subdural space, its removal is a matter of much more moment. To remove it from the pia, in which case it is usually firmly adherent to the cord, is by no means easy, and usually means some damage to the superficial layers of the cord. The patient receives no immediate relief from such an operation, and may be even worse for a time, owing to the destruction of superficial paths and postoperative edema. Nor is the course of the disease necessarily checked by the procedure; for a new layer of scar tissue may form after the operation, just as we know it to form after the excision of a keloid, and the like.

The operative treatment of fibrous meningitis is, therefore, very

dubious, and if its diagnosis could only be made to a certainty, it would not be operated upon.

Granulomata.—These lesions, save in rare cases, will be either gummata or conglomerate tubercles when seen at operation and lie intradurally. Extradural tuberculosis means tuberculosis of the vertebræ, and is usually best treated by orthopedic measures. Syphilis, outside the dura and producing cord symptoms, is rare, and needs no operative treatment. If a granuloma be found inside the dura, the diagnosis will not be made, as a rule, between syphilis and tuberculosis, unless it has been made previous to operation. Of course, the greater frequency of caseation in a tubercle and the greater vascularity of a gumma are differential points of some importance. If the lesions are on the dura or pia, the best thing to do is to wash out with a sponge wet in sublimate, 1 to 1000, somewhat as Horsley does in the cases of brain syphilis. Some good has been done by the sublimate, and by the decompression, and no essential harm is done. If, however, the granuloma be tuberculous, the patient is in immediate danger of developing an acute tuberculous meningitis. The dural space should be well walled off with iodoform gauze and all caseous material washed away with 1 per cent. carbolic acid, followed by normal salt solution.

The same objections hold for the operative treatment of conglomerate tubercles of the cord. Such a tubercle may be extruded after the longitudinal incision has been made in the cord, but it will probably only result in an acute fatal disease, namely, tuberculosis of the meninges, instead of the more prolonged course as a focal disease of the cord. A gumma, of course, if accurately diagnosticated, could probably be removed by extrusion with very little danger to the meninges, especially if preceded and followed by local irrigation with sublimate, and a vigorous treatment with mercury rubs or salvarsan, or both.

The treatment of granulomata of the cord is by no means so grateful a field for the surgeon as when they occur in the brain, and even then the results, so far obtained, are only fairly encouraging, owing to the frequent presence of other "latent" foci and subsequent acute disseminations.

Acute Inflammatory Processes Causing Pressure on the Cord.—Acute inflammation inside the dura usually means death to the patient from meningitis. Lumbar puncture is our diagnostic procedure, and practically the only surgical measure of any importance.

The extradural processes are the ones with which we are chiefly interested here. They proceed, as a rule, from the bone as acute osteomyelitis, either primary or secondary to pyemia. The diagnosis is difficult, because of the slightness of the local symptoms and the rarity of the lesion. We have seen such a case at *post mortem*, but have never had the opportunity to treat one *intra vitam*.

Should the focus lie in the neural arches or spinous processes it would be relatively easy to attack. When it lies in the body of the vertebra it is very difficult to get at, and the dangers to the cord and roots are very considerable. The x-ray, unfortunately, in these acute cases

gives us practically no assistance in locating the lesion, and any surgical procedure in this connection is largely exploratory.

Resection of the Posterior Nerve Roots.—This procedure has been recommended and performed for (1) pain in the region of distribution of the nerve roots; (2) the visceral crises of tabes; (3) for muscle spasm, particularly when due to disease of the upper motor neurone.

The procedure is by no means new, but has recently sprung up and achieved considerable vogue in many quarters. Robert Abbé, of New York, who performed this operation for the first time, gives the credit of its conception to Dana, on whose patient the first operation was performed. Foerster in his address before the Royal Society of Medicine, June 13, 1911, admits this priority. There seems to be, therefore, no valid reason why Americans may not with pride call this the Dana operation, or at most the Dana-Foerster operation, in recognition of the important role which Foerster has played in the recent extension and popularization of the procedure.

Dana's original case was operated because of a neuralgia of branches of the brachial plexus. In the following year, 1889, Abbé had another similar case. Sir Victor Horsley had two cases of severe neuralgia of the arm in 1888, but somewhat after Dana's first case. Chipault, slightly later, reported five cases of posterior root resection for neuralgia of the arm. Giordano operated a case of sciatica, and Faure an inoperable case of carcinoma of the uterus, in order to relieve the neuralgic pains.

Foerster did the first resection of the posterior roots with Tietze, in three cases of metastatic carcinoma of the spinal column with severe nerve-root pains. The results were only temporary, however, because the advancement of the disease soon involved other roots.

In 1900 Mingazzini proposed resection of the posterior roots for tabes with severe lightning-pains and gastric crises. Hildebrand, in 1910, was the first to perform the operation for this indication. He cut two cervical roots. The result obtained was only temporary. Enderlen was the next to try the operation. He cut some of the thoracic roots.

Gottstein and Foerster then had a case on which they cut the fourth and fifth lumbar and the first sacral roots on account of tabes with a localized neuralgia of the internal malleolus. The effect of this operation was only temporary.

Since Foerster's early publications the operation has been taken up by many different operators. Of the various indications for which the operation is performed, perhaps the least successful indication has been that of tabetic crises. In the cases in which it was done chiefly for the pain of the crises, the operation has been a success in 30 to 60 per cent.; but the crises in which vomiting is the chief feature have received distinctly less relief. Why all cases are not successful it is difficult to say. The case of Guleke throws some light on this question. In a patient on whom he performed resection of the seventh to ninth roots, the crises disappeared for a time. They recurred but again disappeared after the resection of the tenth and eleventh thoracic roots.

This case would appear to show that our usual resections are not extensive enough, because of the large number of roots carrying sympathetic fibers to the gastro-intestinal regions. Although to increase the number of roots resected increases the gravity of the operation, Foerster suggests the advisability of resecting as high as the fifth or sixth and as low as the eleventh or twelfth roots instead of removing only the seventh to ninth, as was done at first.

When the lightning pains are in an extremity, Foerster recommends the cutting of all the roots to that limb. Taylor, on the other hand, considers that by cutting *all* the roots, but not completely through, a sufficiently satisfactory result may be obtained. That the sensory defects, after such resection are often surprisingly small, is a fact that has excited the wonder of many observers. Sherrington's observation on monkeys that the cutting of less than three consecutive roots produce no demonstrable defect, seems to be the case in man to perhaps an even greater extent.

In Foerster's first case, in which he cut the fourth and fifth lumbar and first sacral nerve roots, there were no sensory changes demonstrable in the leg. If every other root be cut over a considerable distance, the sensory changes are practically *nil*, perhaps scarcely even a change in the acuteness of sensation being demonstrable. Of course, as a rule, such minute and painstaking examinations as were done, for example, by Rothstein and Hamill, on their syringomyelia patients, examinations taking months of time for completion, have not been done on many, if any, of these cases. But to all ordinary observers sensation is practically intact.

Again, it may be that the pains in many of these cases are entirely central in their origin, and are, therefore, not susceptible of relief by any measure applied peripherally. Then, again, crises may contain a pneumogastric factor, especially those associated with severe vomiting. In such cases Exner's operation, resection of the branches of the pneumogastric nerve below the diaphragm, according to his technique, or even above the diaphragm, by the transthoracic operation, would be the logical procedure rather than the nerve-root resection. This subject is, however, still too new to permit of any thorough discussion.

Borchardt has condemned it on an experience of one case only.

Most important in order to determine just which roots to resect is the exact localization of the pain, the extent of the hyperesthesia of the skin, and the site of its maximum intensity. The segment to which this cutaneous area belongs should then be taken as a middle point among the roots to be resected. In cases of gastric crises without pains these localizing factors are not present, and therefore, in such cases, we believe the operation should not be performed.

Value of Resection.—The value of resection of the posterior roots in spastic paralysis due to disease of the upper motor neurone is somewhat greater than in the case of tabetic crises or neuralgic pains. But to achieve the best results these cases must also be as carefully selected as the tabetic cases. The upper motor path carries two kinds of fibers,

one bearing the motor impulse, injury to which causes paralysis or paresis of the muscle supplied, the other carrying inhibitory fibers which check the sensory stimuli constantly entering the spinal cord by way of the posterior roots. It is the damage to the latter class of fibers which causes the spastic symptom-complex, because of the loss of control over the inflow of sensory impulses into the reflex arcs of the cord. If, now, the posterior roots be cut, the sensory impulses are again reduced, and spastic symptoms are diminished or disappear. Frequently the fibers carrying motor impulses are fairly intact when the inhibitory fibers are practically out of commission. In such cases resection of the posterior roots is followed by complete disappearance of the spasms, and therefore all those voluntary movements which were interfered with by the spasticity are now executed without difficulty. Success can be anticipated only in those cases in which voluntary control of the muscles is still present to some extent, and when the spasticity is due to the influx of excessive sensory stimuli in the reflex arc in the gray matter of the cord. Therefore, the spasms of athetosis and chorea offer no prospects of success, because these conditions are due to the pathological stimulation of motor centres and paths lying in the cerebrum and midbrain. Resection of the posterior roots does not alter these pathological stimuli descending from above.

Indications.—The class of cases in which the operation is indicated, embraces (1) the infantile cerebral palsies (Little's disease); (2) spastic spinal paraplegias which may be traumatic, due to Pott's disease, syphilitic spastic paraplegia, primary spastic paraplegia of Erb and Charcot, disseminated sclerosis, and Parkinson's disease.

In general to justify attempting the operation, there must be a reasonable prospect that innervating fibers of the pyramidal tract are still present, and, therefore, all cases with slight spasm and much paralysis are, as a rule, unsuitable. In cases of severe spasm and total voluntary paralysis there may remain a considerable amount of voluntary excitability only apparent after the elimination of spasm. In such cases the experiment is well worth trying. A preliminary injection of stovain or novocain may be made in order to decide whether any voluntary motility remains. The anesthetic temporarily removes the spasticity.

The arm, as a rule, is less suitable for the operation than the leg. Whatever the lesion back of the condition, it must be practically stationary in order to insure a satisfactory result. A progressive disease, like multiple sclerosis, is hardly suitable for the operation. Little's disease, traumatic cases, and old syphilitic paraplegias give a much more favorable outlook.

Permanent good results can be obtained only where the operation is followed by careful and prolonged after-treatment, consisting of exercise, massage, and mechanical and orthopedic measures. It is particularly by exercise that voluntary motility is brought up to its highest level and only then does standing and walking become impossible. Such exercises should often be continued for years. When retention

in casts is necessary to correct the previously existing deformity, the limbs should be removed from the cast daily for the purpose of giving massage and going through the exercises. When organic contractions, due to the shrivelling of muscles and tendons are present the usual tenotomies and other orthopedic measures should be taken.

As to the technique of the operation, it is simply that of laminectomy with or without subsequent opening of the dura in order to get at the roots. Dana originally proposed to open the dura, and Foerster prefers that method. It simplifies the operation, although it is often more difficult to distinguish the different roots.

Guleke and others prefer the extradural excision because of the greater ease of identifying the roots and the absence of danger of meningitis. Mr. Hey Groves' introduction of the use of adrenalin to control the hemorrhage may sometimes be of use, although we have so far found the compression with hot towels perfectly adequate in our laminectomies.

The unilateral laminectomy for root resection, as proposed by Taylor, we have had no experience with. If it is anywhere useful it would be in cases of root resection. We prefer, however, the large free field of vision and believe that it is better to cut away a little extra bone than to subject the patient to the danger of having an operator working in the dark on so important and delicate a structure as the spinal cord.

Secondary Operations of the Cord.—In such cases, in which the spinal processes and the arches have been cut away the chief difficulty is the absence of landmarks to guide us in cutting down on the dura and cord. The dura is, as a rule, closely adherent to the muscle and scar tissue masses lying directly above it. Therefore, if we use the scar from the previous operation as a guide on which to cut down, we must use the greatest care that in doing so we do not cut through the dura and even incise the cord. The tissues must be cut layer by layer just as in the cases of laparotomy when we fear to cut suddenly through the peritoneum and thereby injure the gut. We are especially careful always to have our knife cuts perfectly longitudinal to the long axis of the body. Then we are the least likely to do damage to the cord in case we should accidentally incise it.

A safer, if somewhat longer, procedure is to start the incision at one end of the previous wound and cut down on an intact arch, bite this away with the forceps, and open the dura. Once the dura is opened, we can reopen the old incision by cutting down on a grooved director placed in the subdural space as a guide.

If it is not desirable to open through the old scar, a lateral incision may be made to one side of the old scar and continued downward to impinge on the lateral remnant of one of the arches. This remnant is then bitten away with the rongeur forceps, the dura incised from the side, and the cord exposed by dividing the soft tissues on a grooved director or similar guide.

While it is not often necessary to perform secondary operations, yet recurrence of tumor symptoms, whether due to actual recurrence of the tumor or to the formation of a localized meningitis serosa, failure

of a previous operator to find a tumor which subsequent developments show must be present, second-step operations for the removal of cord tumors, extruded after preliminary incision in the cord and the like, do sometimes compel its performance. It is then an ungrateful though necessary task.

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Filed by Preservation 1999